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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE ETIOLOGY AND PATHOLOGY OF LOSS OF VISION FROM THE ACCESSORY SINUS.*

DR. LEON E. WHITE, Boston, Mass.

So far as can be determined, from discussions with men having to do with these border-line cases, the opinion almost universally held is that accessory sinus blindness pre-supposes purulent infections. To illustrate: In a recent conversation with a noted cerebral surgeon who has given this subject considerable attention, he expressed his conviction that if loss of vision ever resulted from the sinuses it would only occur when they were most evidently diseased. When mention is made of opening them for the relief of blindness, the first question almost invariably is, "Did you find pus?" While cases have occasionally been reported of loss of vision from non-purulent infections, this old belief of the necessity of finding pus is so persistent that it warrants reconsideration.

ETIOLOGY.

The earlier writers dwelt largely on the anatomical relations of the optic nerve and gave undue emphasis to the theory that purulent infections in the sinuses spread to the optic nerve because only a very thin bone intervened.

Gradle's views of the etiology of blindness shows a beginning reaction from this idea. He says:

"I believe that I am justified in stating" (contrary to the teachings of Onodi and Loeb) "that the anatomic relations of the sphenoid

*Read before The American Laryngological, Rhinological and Otological Society at Atlantic City, N. J., June 3, 1921.

and ethmoid cells to the optic canal are immaterial when it comes to a question of optic nerve involvement in accessory sinus disease. The trouble is transmitted by the soft tissues alone."

In my first cases I expected to find the sinuses filled with pus, but this happened so rarely that it only helped to explain the etiology in but a few cases.

I did not at that time realize how difficult it was to determine histologically just where the dividing line was to be drawn between suppurative and non-suppurative conditions. Even when pus had been previously seen in the nose, it was found to be extremely difficult to detect it during the operation, mixed, as it necessarily must be, with blood. In not a few cases where pus was not seen, either before or at the time of operating, the slides would frequently show dead cells, detritus and cocci in the lymph channels.

When Sluder's masterly work appeared I thought hyperplasia might explain the etiology of many cases, as it probably does when extreme. But it has not been my good fortune to find enough macroscopic hyperplasia to warrant it being considered the *principal* cause for the loss of vision. Nor did the sections, when viewed under the microscope, show changes sufficient to so consider it, although Dr. Jonathan Wright aptly says on this point:

"Are you or is any man in a position to state definitely just how much tissue change is sufficient to cause impairment of vision? I am sure I never was. I think it reasonable to suppose that is more or less dependent on locality, individual configuration of the nasal chambers and the sinuses and many other factors, not forgetting the general one, which is systemic and undefinable. No factor, not even 'tissue change' or obstruction is ever the sole etiological factor in any disease anywhere."

Being, then, at a loss to explain the etiology, I went carefully over my cases and discovered that the one almost constant finding was enlargement of the turbinate tissue adjacent to the sphenoidal ostium. It was also quite usual to have a posterior deflection of the septum crowding the already poorly ventilated regions of the posterior sinuses. Then, in operating, the superior turbinates were generally found to be markedly enlarged and it was necessary to take out more tissue than one would have thought necessary from previous inspection before good exposure of the wall of the sphenoid was obtained.

While Onodi did not recognize the full importance of blocking to ventilation and drainage, he has this to say in his explanation of the importance of pressure by the large cystic turbinates:

"The so-called bone cyst is a structural anomaly known as the turbinate cell. Diseased, these turbinate cells may attain various sizes. They appear in the superior and middle turbinates, and, as the turbinate cell of the superior turbinate, according to our observations, may be in close relation to the optic nerve; it will be seen that the practical importance of the turbinate cell is due to pathologic changes and growth, especially through pressure.

"In cases under our observation the turbinate cell appeared nine times in the middle and twice in the superior turbinate; the turbinate cells twice opened into the middle meatus, at other times in the superior. Its length varied from 8 to 20 mm., the width from 5 to 14 mm., the height from 7 to 16 mm."

Thus, in reviewing my cases it seemed that the only consistent explanation of the etiology, applicable to most of the doubtful ones, was to be found in the size and position of the middle and superior turbinates.

The following extracts are taken from the published reports of these cases and show the unconscious evolution of this theory. In the earlier ones little attention was paid to the middle turbinate, in a few they were not mentioned, but on looking back I feel there was some obstruction in practically all. Abstracts from the records of three other cases (5-7-20) are also given, while noted at the time, did not then appear of sufficient importance to be incorporated in the published reports. The last 12 cases all indicate how important this matter is now considered.

Case III: "On examination there was found on the left a moderate deflection of the septum and an enlarged and swollen middle turbinate completely blocking the superior meatus. * * * Right middle turbinate was swollen, but not as firmly wedged in as the left."

Case VI: "Septum deflected to left (the eye affected) and middle turbinate greatly swollen. * * * Posterior portion of left middle turbinate was obstructing the opening to the sphenoid."

Case IX: "Right middle turbinate tightly wedged between septum and ethmoidal wall."

Case XII: "A posterior deviation of the septum crowded the left middle turbinate so as to obstruct the sphenoid and posterior ethmoid."

Case XIII: "Marked deviation of the septum and hypertrophied middle turbinates."

Case XIV: "A slight deviation of the septum high up and both middle turbinates were greatly swollen blocking the posterior ethmoids."

Case XVI: "Marked deviation of the septum to the right and the middle turbinate tightly wedged between it and the outer wall."

Case XVII: "Both middle turbinates were greatly hypertrophied, the left the most marked and especially obstructing the sphenoid."

Case XVIII: "The septum was deflected to the opposite side, but the turbinate on the affected side was enlarged and especially obstructive."

Case XIX: "Marked hypertrophy of the middle turbinate on the affected side evidently obstructing the accessory sinuses."

Case XXI: "Both middle turbinates were enlarged and obstructing."

Case XXII: "The middle turbinate on the affected side was enlarged and obstructive."

Case XXIII: "The septum was deflected; and while the anterior end of the right middle turbinate was small, the posterior portion was large and obstructive."

Case XXIV: "The right middle turbinate was somewhat obstructive posteriorly. The middle turbinate which proved to be much larger than it appeared, was removed."

Case XXV: "The posterior portion of the left middle turbinate seemed somewhat enlarged."

Case XXVI: "Nasal examination disclosed an enlarged obstructive middle turbinate."

Case XXVII: "A slight deflection of the septum to the left which extended far back, crowding somewhat the large unhealthy middle turbinate on that side. The right middle turbinate was also enlarged, soft to the touch and about it there was considerable secretion. Both middle turbinates without doubt obstructed the posterior sinuses."

Case XXVIII: "Nasal examinaion revealed marked deflection of the septum to the right, but the turbinates did not appear especially hypertrophied or obstructive." (This case recovered without operation.)

Case XXIX: "Nasal examination revealed inflamed middle turbinates. They shrunk, however, under cocain to the normal size and as they did not seem to be especially obstructive, the prediction was made that recovery would take place without operation." (Recovery did so take place.)

The following notes on these *three* cases did not appear with the reports, but are taken from my records:

Case V: "Examination of nose revealed a somewhat deflected septum to the right, and middle turbinate was rather crowded."

Case VII: "Hypertrophy of middle turbinates."

Case XX: "Right middle turbinate considerably enlarged and reddened. Cotton swabs, however, could be placed between it and ethmoidal wall (so that it was felt case would recover without operation, which it did)."

The following *four* cases, which appear at the end of this paper, are added to make the record more complete:

Case XXX: "Septum deflected to right posteriorly, crowding an enlarged middle turbinate and evidently blocking the posterior sinuses. Left middle turbinate also moderately hypertrophied."

Case XXXI: "The right middle turbinate was somewhat enlarged and the septum bent slightly to that side."

Case XXXII: "The septum was badly deflected high up with enlarged middle turbinates in close contact with it."

Case XXXIII: "A moderately high posterior deviation of the septum to the left in contact with a somewhat hypertrophied middle turbinate. There appears to be considerable obstruction to the drainage of the posterior sinuses."

To Summarize: In the last 22 cases, mention is made of obstruction by the middle turbinate in all but 2. In the first 10 it is mentioned but 5 times, as little of importance was at first attached to it.

Thus, the frequency with which these middle and superior turbinates have been found hypertrophied and most evidently blocking the posterior sinuses suggested the following theory as to etiology, in many cases where pus cannot be detected and the roentgenograms are practically negative. While it may be premature to present this theory without more data than I have been able to secure, it is done to obtain your views as to its plausibility and to solicit your co-operation in accumulating proof of its correctness.

The theory, in short, is that these superior turbinate structures high up in the roof of the nose, gradually become enlarged and obstructive. There is an increase in the basal connective tissue, *i.e.*, a certain amount of hyperplasia, so that eventually the posterior sinuses are practically blocked. Then some infection takes place in these sinuses which, if not already occluded by the hypertrophied turbinates, most readily become so by a very slight increase in their size. Thus, a low grade inflammatory process commences, generally of the exudative but non-suppurative type, the swelling and infiltration incident to it spreads by continuity of tissue to the optic nerve, or if the sinuses are completely shut off, there may be a closed

empyema with infection to the nerve through the circulation,—not in all cases, of course, but in those which, through some anatomical peculiarity, are rendered especially vulnerable. The infection may be confined to the region superior and posterior to the middle turbinates, thus explaining why so little is seen on inspection and why roentgenograms show but slight, if any, changes, *i.e.*, only what would be expected from an acute non-suppurative inflammatory process. The examiner should visualize these conditions somewhat, as it is not possible by any method of description to tell exactly how to determine these turbinate abnormalities. It is comparable to the difficulty of expressing to others why, in palpating the two mastoids the skilled otologist can tell that the one is filled with pus, with cellular structures broken down, and the other normal. It is simply the little edema that to the experienced examiner means so much, and yet it is so slight that only by long practice can one be reasonably certain of his diagnosis.

In my efforts to state a simple understandable theory, it is impossible to consider the manifold variations and the many exceptions that one is bound to meet in these cases. Nature is complex, but the mind of man seeks repose in a simplicity of cause and effect which does not usually exist.

As to whether or not the damage to the optic nerve occurs secondary to involvement of the sympathetic system in this region, as Dr. W. H. Haskin believes, I am unable to say. His theory seems a good one and the following is quoted:

"The fibres of the carotid and cavernous plexuses are in intimate contact with these areas (diseased cells or sinuses) and they are the vasomotor and trophic nerves, the pupil dilating nerves and those which control the unstriped muscles of the eye and the orbit and they all belong to the sympathetic nervous system."

PATHOLOGY.

Having meditated much upon the pathology and discussed it, or the lack of it, not a little with many of my confreres without especially satisfactory results, the conclusions I have drawn are so similar to those in the following paragraph that I will read it:

"Sidepaths in Medicine," by Jay Perkins, M. D., Providence, R. I.—"The study of pathology is an absolute necessity to one who would have a knowledge of medicine, but if we get into the habit of thinking a disease must present the pathology as taught and seen in the autopsy room before we make a diagnosis of that disease, we are many times of very little use to the patient, because we

fail to recognize the nature of their ailments while they are yet in a curable stage."

It is well to bear in mind that noses filled with polypi and pus rarely produce any effect on the vision. It is also well to remember that cases with multiple sclerosis, brain tumors and various constitutional conditions may incidentally have marked pathology in the nose, so that no matter how much one sees on inspection, other possible causes should always be considered. The classification I have tried to make is:

1: Direct extension by continuity from the source of infection to the optic nerve;

2: Toxemia from some infective process;

3: Bacteremia or focal infection; and

4: Hyperplasia.

The theory of *direct extension,* it seems to me, has never received the emphasis it merits. If it is the correct explanation of what takes place in a considerable portion of the cases, it helps to clarify this whole subject. Let me quote Onodi again on this point:

"We have two accurate observations, made by Ortman and Hajek, who have established the cause of infection by way of the blood vessels and tissue continuity. * * *

"In Ortman's case the body of the sphenoid and the membranes showed the presence of diplococci. This finding showed that infection of the meninges may take place by direct continuity. * * *

"The case observed by Hajek showed * * * a diffuse, fibrous, suppurative, acute pachymeningitis interna, and leptomeningitis of the base and convexity of the brain with acute internal hydrocephalus, inflammation of the left ethmoid cells, edema of the mucosa of the maxillary sinuses. * * *

"That infection may take its course through tissue continuity and the blood vessels has been proven; that it may make its way through the lymph vessels, along the lymph spaces of the olfactory nerves through the openings of the lamina cribrosa, is possible, but has not been, up to date, microscopically and bacteriologically confirmed."

The theory, as I conceive it, is that by direct extension an acute inflammatory infection originating in the posterior sinuses, rapidly extends by continuity of tissue to the sheath or even to the optic nerve itself. These infections are frequently non-suppurative with practically negative roentgenograms. An obstructive middle turbinate, and only the posterior portion of that, may be all that is found.

Because it is not always possible to secure sections showing marked changes, many have questioned the possibility of the sinuses

being a factor in any of these border-line vision cases. Why, they ask, do you get loss of vision with little or nothing to be seen in the nose and yet rarely any visual disturbances in those so evidently diseased? The explanation, it seems to me, is that in chronic cases nature usually walls off the eye, the optic nerve and, in fact, the body from the source of infection. With this walling off, the blood also acquires an antitoxic immunity. An antrum, a frontal or any accessory sinus may be filled with the most offensive pus and be lined with exuberant granulations, yet the patient suffers but little, if at all. Toxemia or bacteremia may possibly result, but as a direct causative factor these nasty sinuses which we have all been looking for in acute visual disturbances, play but a minor role.

While Onodi did not seem to take into account this walling off process, he mentions that marked infections do not necessarily impair the vision, as follows:

"We are acquainted with several cases in which, in spite of the presence of suppuration and destruction of the bony walls of the sphenoid sinus, the sight was not impaired. Berger and Tyrmann mention the slow erosion or wearing away of separate parts of the sphenoid body, though no derangement of sight took place, but later meningitis appeared. Baratoux mentions a case in which a large part of the sphenoid body was thrown off through the nose, causing no disturbance whatever. Hajek saw a number of cases of syphilitic destruction of the anterior wall of the sphenoid sinus without special symptoms of any other kind. Flatau reports 26 cases of empyema and caries of the sphenoid sinus, but mentions no derangement of sight. Foucher describes the case of a girl of 15 with necrosis of the turbinates and the sphenoid sinuses; antiluetic treatment was instituted, bone sequestra were formed and the girl died; sight disturbances were not observed. The observations of Schaffer, Onodi, Schmiegelow and Hoffman also show that in many cases of diseased sphenoid sinuses no changes in the function of the optic nerve took place."

In the chronic empyemas, nature generally throws out a barrier which walls them off from the adjacent structures, otherwise purulent infections in the sinuses and likewise in the mastoid would more frequently invade the cranial cavity. While it is true that through some re-infection or a breaking down or erosion through this barrier there occasionally occur serious intracranial infections, these are of rather infrequent occurrence. The otologist well knows how important it is to await the formation of the barrier which separates his mastoid from the meninges and lateral sinus before he advises

operative interference. While he may not be able to demonstrate pathologically what happens in these acute infections, he learns from his clinical experience that during the first week or ten days there is more or less general infection, frequently associated with hyperpyrexia and accompanied by tenderness over and about the mastoid together with more or less pain. Then the mastoid becomes separated from the general system by this walling off process and although its cellular structure may be breaking down, the infection is practically a localized one and pain, tenderness and temperature usually subside.

This is comparable to what may happen in the accessory sinuses. When an acute infection occurs in the sphenoid or posterior ethmoid, there may be an almost immediate invasion of the tissue about the optic nerve, including the sheath of the nerve or the nerve itself. When, on the other hand, there is an infection in a more remote sinus, it fortunately becomes walled off before the optic nerve is reached. Should the infections, however, persist there may be poured into the system a certain amount of bacteria and toxins which may produce an optic neuritis, or, for that matter, a neuritis of any nerve.

What I especially desire to emphasize is that while subacute or chronic infections may be an occasional factor in involvement of the optic nerve, the acute infections with but little discoverable evidence in the nose are of far greater consequence. The fact that it is so easy to overlook this condition leads me to dwell upon its importance.

In but few of my cases was there marked involvement of the sinuses, and in none was there offensive pus. The important thing to look for is obstruction to the posterior sinuses. In my first 10 cases the nasal examination was reported negative in 7, while in the last 17 some obstruction to drainage was found in all. It required these 10 cases to teach me what to look for; and without doubt were they to be again examined I should not pronounce them all negative. Let me emphasize again that normal noses do not produce loss of vision. But in many, that on first inspection *seem* normal, there *is infection*, even if not evident. I think it may be stated in a general way that if the inflammation has simply produced pressure by a swelling about the nerve and has not involved the nerve itself, the prognosis is good; while if it involves the nerve (if there is a true optic neuritis), complete recovery is much less liable to take place. In the latter the sinuses should be drained and drained speedily.

For one should remember that the optic nerve is really not a nerve, but a lobe of the brain. It is easily destroyed and does not regenerate. Parsons says of it:

"The so-called optic nerve, together with certain parts of the retina, constitutes a lobe of the brain, and has therefore the characteristics of the central nervous system. Hence the nerve fibres are devoid of a sheath of Schwann and the interstitial substance is neurolgia."

In a conversation with Dr. Sluder as to the difficulty I was having in obtaining slides which showed much pathology, he told me how much help he had received from Dr. Jonathan Wright and suggested that I write to him, which I did, and his kindly letters have so many points applicable to this subject that I have asked his permission to make extracts, justly feeling that it would be selfish not to share the wisdom therein contained. In one letter written in reply to my inquiry as to the best methods of investigating the pathology, he says:

"The reasons the general pathologist fails to recognize the basis of much nasal clinical manifestation, is that it frequently happens that the tissue submitted is too small for a definite opinion, or is from a locality not directly associated with the trouble—for instance, it not infrequently happens that the nasal surface of the sphenoid is fairly normal and that of the cavity, diseased—the tissue may be from the middle turbinate rather than from a sinus wall, etc. Chiefly, however, it is because the phenomena of physiological change are so confused with those of the pathological. There is considerable difference in even the normal structure, dependent on age and season or clime, but next to the one I have emphasized, probably the most frequent practical difficulty comes from the ravage wrought in the process of getting rid of the bone salts to allow sectioning."

In another letter he says:

"I do not think I realized how impossible it is for the general pathologist to follow the bone changes in the nose intelligently until I began to go over Sluder's work, but it is now quite evident that at least, so far as the nose is concerned, that objective study of tissue changes in the chronic inflammations must go hand in hand with clinical study. In fact, that is the only rational way to study inflammations anywhere. When a man has a piece of tissue brought to him and has no first-hand knowledge of the dynamic processes which have put that tissue in its condition, how can he give the clinical man what he wants? He can say if a thing is cancer or not, but in chronic inflammations he has a static condition thrust upon his attention when really it is a process which the man, bringing it to him,

wants to know about. In the nose no man alive or dead can or ever could tell where the normal leaves off in adults and where the abnormal begins. It is difficult enough for the conscientious clinical observer of the process in situ (the man who sees the thing going on in a patient with whose general condition he is familiar), it is hard enough for him to say whether it is really a menace to his patient's health or his comfort, but what can the pathologist tell him with looking at his bit of tissue in a matter where the so-called abnormal process is merely a continuation, exaggerated if you will, of normal physiological development or retrocession of erectile or lymphoid tissue? It is almost instinctive groping after lines of differentiation which exist no more here than between other artificialities erected by man for the help of his puny powers of comprehension, landmarks for his memory, the line between inflammation and the turgor of physiological growth, the line between physiological and pathological, whether of the lymphoid tissue in the throat or the erectile tissue in the nose. The laboratory man knows little or nothing of the looks of the nasal chambers in childhood, in adult life and in old age, and the very greatest differences are familiar to us by virtue of our daily observations in the clinic. What is plainly abnormal in one age may not be so at all in another. But these are gross distinctions we are familiar with as clinicians."

Possibly by comparing what happens to some of the other cranial nerves enlightenment may be gained on the pathology in these vision cases. There is a not infrequent paralysis of the facial nerve from some slight disturbance in the middle ear. This is generally known as Bell's-palsy. Reik claims there is always a preceding non-suppurative middle ear infection, yet it is of such a slight or transitory nature that the drum membrane appears practically normal. The fact that both the facial and optic nerves pass through a bony foramen and are thereby rendered more susceptible to inflammatory affections, would seem to make this comparison of value. When there is an inflammation of the middle ear sufficient to cause a facial paralysis and yet not active enough to produce much, if any, change evident even through a *transparent* drum membrane, *why* expect to discover in the posterior portion of the nose a change *so marked* as to be evident through the *mucous membrane covered walls* of the accessory sinuses, not to speak of the difficulty of inspecting these walls owing to the presence of a middle turbinate? The middle ear may well be compared to the sphenoid, the eustachian tube to the ostium. When closed an inflammatory process takes place sufficient in the one to cause facial paralysis; in the other blindness.

Let me read a few extracts from Reik. In an article on the relationship between otitis media (non-suppurative) and facial paralysis of the refrigeratory type, he says:

"In the majority of cases, if not indeed in all, of facial paralysis of the refrigeratory or rheumatic class, an acute or sub-acute otitis media is an intermediary condition between the exposure to cold and the appearance of the paresis."

He quotes Dr. Herter's opinion as to the pathology as follows:

"Neuritis of the facial nerve is a cause of a very large proportion of all cases of peripheral palsy. The neuritis is commonly called rheumatic, whatever that may mean. It is true that the palsy generally comes on after exposure to cold. Occasionally the subjects are distinctly gouty or rheumatic, but usually there is no evidence of either condition. It was formerly thought that exposure to cold caused facial paralysis by paralyzing the terminations of the nerve in the facial muscles. There is now good reason to believe that the infection always depends upon an inflammation of the trunk of the nerve (perhaps involving chiefly the sheath) within the Fallopian canal, and there is no evidence that inflammation ever involves the nerve after it emerges from the canal. * * *

"The facial nerve is vulnerable only in that portion of the Fallopian canal which extends in close proximity to the middle ear. The Fallopian canal is separted from the middle ear by an extremely thin bone which frequently shows dehiscences so that any inflammatory condition of the middle ear readily extends to the sheath of the facial nerve. * * *

"Patients with Bell's-palsy generally have a history of some slight disturbance in the ear a day or two before the onset of the paralysis, but *many of the ears have cleared up so that they appear practically normal on inspection.*"

In his text-book on "Diseases of the Ear, Nose & Throat," published in 1911, Reik says:

"In every case of refrigeratory facial paralysis the ear should be immediately and carefully examined and if there is the slightest reason for supposing middle ear trouble, the tympanum should be opened at once. * * * In fact, the author is inclined to urge tympanotomy in every case at the earliest possible moment, even when you cannot secure visible evidence of intratympanic inflammation, because, as has been pointed out, there probably is disease there which is not clearly perceptible. A clean incision through the tympanic membrane can do no harm if there is no disease there and the wound will heal without causing any damage to function."

As these references were rather old, I wrote to Dr. Reik as to his present views, and his reply on January 13, 1921, is as follows:

"In regard to my views upon refrigeratory facial paralysis, I candidly say that they have not changed in any sense since the publication of the articles you refer to.

"In every case of a refrigeratory facial paralysis that I examined after expressing the views you refer to, I was able either to demonstrate the existence of acute or subacute middle ear inflammation, or to show by the hearing tests that there was an abnormal middle ear condition, even though there were no visible signs of disease. Paracentesis and middle ear treatment certainly facilitated materially the progress of these cases.

"I think the comparison that you draw between this condition and visual defects, possibly due to accessory sinus disease, is quite logical. The explanation in both cases may lie in an irritation or inflammation of the nerve sheath, with resulting sensory disturbance, without the actual presence of pus or infective material in the adjacent cavities. Operation is certainly justifiable under the conditions that you suggest."

There is one other cranial nerve—the 6th—that occasionally becomes involved in an inflammation of the mastoid. This nerve also passes through a canal and although a fibrous one, it is just where the nerve is vulnerable. The following is taken from Perkins' article, "Abducens Paralysis & Otitis Media Purulenta" (Annals of Otology, 1910):

"The cause of the occurrence of abducens paralysis and the key to its solution will be found in the peculiar anatomic arrangement by which the sixth nerve gains its position in the outer wall of the cavernous sinus and comes into relation with the ophthalmic division of the fifth, the fourth and the third cranial nerves, which it does immediately anterior to the first intracranial portion of the carotid artery. Anterior to this, there is no special reason why one of these nerves should be more frequently involved than the others. Posterior to this point, however, the sixth nerve is comparatively isolated and passes through a fibrous canal, called 'Dorello's Canal' * * * which begins where the nerve pierces the dura in the posterior fossa. * * * Now it is evident that narrowing of this canal may result from inflammatory thickening of its walls, or this thickening may arise from edema, caused by inflammation in the neighboring dura or bone. This narrowing, however produced, would result in pressure upon the nerve and abducens paralysis; or the inflamma-

tion may extend to the nerve itself, *causing neuritis with consequent loss of function.*"

Toxemia: It is conceded that retrobulbar and optic neuritis can be caused by alcohol, lead, tobacco, quinin, optochin, ethylhydrocuprein, arsenic, lues, etc., so that reasoning by analogy there is little doubt but that toxins originating in the accessory sinuses, or for that matter, anywhere in the body, may have similar action on the optic nerve. The onset is usually less violent than where there is a direct extension and is more apt to be a causative factor in chronic types. There is generally sufficient time in these subacute or chronic cases to make a careful differential diagnosis, and here roentgenograms may prove of great assistance.

In the 33 cases studied toxemia was considered the chief factor in 8. In 4 (8 and 9), diseased teeth were extracted, and the antra opened in 3.

It is quite generally conceded that any *pus focus* within the body may be a factor in these vision cases, and while this paper deals only with the accessory sinuses, other possible sources must not be overlooked.

Bacteremia: It has been demonstrated by Billings that infectious micro-organisms may be carried in the blood stream or by the lymph channels from the foci of infection in the teeth, tonsils and accessory sinuses to the terminal blood vessels in various regions of the body. He has shown how the inoculated blood vessels become more or less occluded by endothelial proliferation and leukocytic infiltration and that the bacteria escape through the vessel walls into adjacent tissue, so that there would seem little doubt but that bacteria within the accessory sinuses may also travel via the blood stream and lymph channels to the optic nerve. Why are we so greedy to accept diseased tonsillar crypts as a cause of arthritis of the great toe, but so slow to accept the view of involvement of the optic nerve in a process so contiguous to it as accessory sinus inflammation? Billings' explanation of the modus operandi in focal infection is so good and so applicable to the subject under consideration that I am led to quote the following extracts from his book on this subject:

"When the body is invaded with pathogenic bacteria the natural defenses are increased by their presence in the tissues and blood. The processes are: first, the phenomenon of positive chemotaxis with resulting leukocytosis and the accumulation of the leukocytes in the areas of infection of the tissues by the formation of local exudates, liquid (purulent) and fibronoplastic, which may serve as walls of protection against further direct invasion; second, leukocytic

phagocytosis with destruction of the invading bacteria; and, third, the formation of protective antibodies in the blood and tissues. * * *

"Undoubtedly the latent pathogenic bacteria usually present in the nose and throat may acquire coincidently with exposure (to cold or following some physical or mental exhaustion), specific pathogenicity, and are able to invade the host because of the lowered resistance and because of added virulency. (There is an) acquisition of specific pathogenicity and tissue affinity by the members of the streptococcus-pneumococcus group. * * *

"Usually a focus of infection is disregarded by the patient and physician unless it cause local discomfort. When a systemic disease occurs which present-day knowledge associates with a primary infectious focus, the site of the focus must be located. The character of the systemic disease may point to the most likely location of the primary portal of infection. The primary focus of acute rheumatic fever, endocarditis, chorea, myositis, glomerulonephritis, peptic ulcer, appendicitis and chronic deforming arthritis, as examples, is usually located in the head and usually in the form of alveolar abscesses, acute or chronic tonsillitis and sinusitis. * * *

"Systemic infection and intoxication from a primary focus is usually hematogenous. The bacteria may be compared with emboli loosened from the place of origin and carried in the blood stream to the smallest and often terminal blood vessels. If virulent and endowed with specific elective pathogenic affinity for the tissues in which they will lodge, and if in sufficient number, the invading bacteria will excite characteristic reactions in the infected tissues and a sequential train of morbid anatomical lesions. The evolution of the anatomical lesions and the clinical phenomena aroused thereby are dependent on the type and virulence of the bacteria, the character of the tissue and the function of the organ involved. The specific tissue reaction consists of a local inflammation with endothelial proliferation of the lining of the blood vessel with or without thrombosis; blocking of the blood vessels; hemorrhage into the immediate tissue; positive chemotaxis with resulting multiplication of the leukocytes and plasma cells in the infected area, or fibrinoplastic exudate with local connective tissue overgrowth. * * *

"Hematogenous focal infection of the nervous apparatus, involving the gasserian and posterior spinal root ganglia and spinal cord, affords confirmation of the infectious nature of herpes, of insular sclerosis and myelitis of the spinal cord. Removal of the primary etiologic foci of infection about the upper air tract and mouth may modify favorably the course of the spinal cord infection,"

and if spinal cord infections, *why not,* let me add, similar infections of the optic nerve?

Hyperplasia: By this is meant a functionless overgrowth of the supporting tissue. It has been greatly emphasized by several writers as a causative factor and in the summary of one of my papers is given as the predominating lesion in 14 cases, but as it is found to be a common condition in the noses of most people subject to the sudden variations of temperature, it is so difficult to explain just what occurs that but few will concede to this hypothesis.

Hyperplasia as a predisposing factor is probably of considerable importance. It undoubtedly renders the accessory sinuses more vulnerable, as was mentioned in speaking of the new etiological theory. Hyperplasia *plus* infection and direct extension to the optic nerve, is, it seems to me, of far greater importance than the mere fact that the tissue has become hyperplastic. In my 14 cases I think it was the infection that caused the damage rather than hyperplasia.

As to the *modus operandi,* Beck believes that "in the early non-suppurative chronic form of sinus disease, there results an engorgement of the optic nerve, edema and later reparative inflammation, contraction and destruction of the fine neural filaments." While no one knows what actually takes place, this is as plausible a description as any I have seen.

Whatever may be the true explanation of the pathology, it would seem at the present time advisable to recognize the clinical fact that where no other cause for the neuritis can be discovered, the opening of the accessory sinuses either by depletion or by ventilation, or by getting rid of some bacteria, generally proves beneficial. Meanwhile, any patient who has been thoroughly investigated should not be deprived of the benefits of this operation just because the bacteriology or pathology is not thoroughly understood.

Let me refer to Loeb's views along this line. In calling attention to the rapidity with which serious eye symptoms may disappear after appropriate nasal treatment, he says:

"The ready recovery under the circumstances is as convincing of their nasal origin as anything could be short of autopsy findings."

While we have not been able to prove definitely to which one of the four classes each case should pathologically belong, enough evidence has been discovered to say pretty definitely that there is an infective process in the posterior sinuses which might readily reach the optic nerve, be it by direct continuity, by toxemia or bacteremia.

Let me here express my thanks and appreciation to Dr. Frederick H. Verhoeff, who, in addition to referring several of these cases

and giving me data in their eye conditions, has also spent much time on the preparation and examination of the specimens, smears and cultures.

Dr. Jonathan Wright very kindly examined the sections from the last nine cases operated upon, and as his report takes up each case more in detail than that of the other pathologists, it is embodied in this paper.

3.3739 Sept. 26, 1919 (Case 19): "I should suppose some of this tissue surely came from an accessory cavity and some from the nasal chambers. The latter is an edematous and fibrous hyperplasia. As to the tissue from the sinus, as I suppose, this presents less edema and more fibrous hyperplasia, a thing in itself significant for the mucosa of a sinus. (I use the term fibrous hyperplasia from long habit as indicating a more chronic process than 'increase of the connective tissue' or 'a large number of fibroblasts,' but perhaps that is not always a warrantable inference.) There is, however, without doubt chronic inflammation of the mucosa and I should say numerous bone areas were involved.

"O.im.1/12—I think confirms this. It seems largely bone proliferation—I am referring only to the bone changes—but there can be no doubt these should be looked on as inflammatory, so marked is the fringing zone of osteoblasts and fibroblasts. Probably no such area of rarefying osteitis can be found here as is shown in the remarkable picture, fig. 4, p. 21, in Sluder's book, but the state of a chronic bone inflammation is not the less unmistakable."

3.3823 Jan. 19, 1920 (Case 23): "Some of the tissue is edematous hyperplasia from the mucous membrane of the nasal chambers themselves with very little connective tissue increase. Narrow strips lined on one side with columnar epithelium and devoid of glands, I should imagine to be from a sinus. These also present the usual appearances of chronic inflammation in the accessory sinuses. I see no bone. Such blood vessels in the mucous membrane of the nasal chambers as are shown perhaps have some increase in low grade connective tissue around them, but show it distinctly gorged with granular detritus and the nuclear remains of leucocytes and new connective tissue cells, probably of lymphocytes too.

"*Chronic inflammation of the nasal and paranasal mucosa.*"

3.3833a Feb. 19, 1920 (Case 24): "The tissue is apparently the lining mucosa of one of the sinuses, but I think inflammatory processes had not been very great or long existing. The cilia of the epithelium are well preserved and I see no bone.

"*Mucosa fairly normal for the nasal sinuses.*"

3.3839a Chron. inflam. turbinate (Case 25): "There are areas of leucocyte infiltration around some of the gland clusters, and perhaps some hyperplasia of the connective tissue, but the elements of the mucosa, epithelium, blood vessels and sinuses, glands otherwise, are not markedly affected.

"*Very moderate chronic inflammation of the erectile nasal mucosa.* Very likely in the middle turbinate areas."

3.3839 Feb. 29, 1920: "Same general character as 3.3839a as to the mucosa, but in the deeper layers here there are some small areas of bone, at whose periphery the cellular activities are not especially exaggerated, I should think. I presume the proximity to the bone here accounts for the general impression of more fibrosis than in the other section."

3.3897 July 29, 1920 (Case 26): "I should suppose some of this tissue was intranasal and some intrasinusodial, or perhaps all of it from near the ostium. The mucosa itself seems to present evidence of acute inflammation, maybe from a prior operative attack, but if not, then in a condition of non-traumatic subacute inflammation. The bone changes, it is less admissible to ascribe to preoperative work, though I suppose this is not impossible in the etiology. However for that, there is a rather marked fibroblastic increase along the margins of the bone areas and the nuclear elements of the bone itself are numerous and deeply stained.

"O.im.1/12: It would take too long to go into detail as to the high-power appearances, but my impression as to the conclusion coincides in a general way with the above, *i.e.*, that we have an inflammation of bone here, possibly due to operative trauma, but if this can be excluded, there seems no doubt of a morbid activity due to other causes." (There had been no prior operation in this case.)

3.4103 March 11, 1921, Turbinate (Case 30): "This is a section through the mucosa and the underlying turbinate bone. All of it shows evidence of a moderate chronic inflammation—hyperplastic. It is pretty well 'mussed up' with the 'decalcifier,' but I should imagine the bone was involved too, though I would not be sure of it."

3.4103: "Sinus mucosa with perhaps some small areas of inflammatory action, but on the whole fairly normal. There is enough new connective tissue to lead to the conjecture that an inflammation was spreading from the nasal chamber in view of the more positive appearance in the previous section, but this is little too fine spun diagnosis to be very reliable, especially in view of the poor showing in the first slide."

3.4103: "Internal wall of sphenoid. More conclusive of inflammatory processes, but one sees less, comparatively, of fibroblastic action and more edema or serous infiltration."

3.4113 (5 slides) March 30, 1921 (Case 31): "(a) Right middle turbinate. Soft parts not especially altered. Some hyperplasia of fibrous tissue near the bone and a very moderate degree of increase in the cellular activity along some bone edges—blood vessel walls somewhat thickened. *Chronic inflammation of deeper elements of the mucosa and of the bone.*

"(b) Another fragment of right middle turbinate. Thinner section—more impressed with bone involvement. Fibrous hyperplasia quite evident.

"(c) Sphenoid. Specimen very small. Rarefying osteitis along some edges rather marked, but soft parts are largely lacking—not very satisfactory.

"(d) Ethmoid. More tissue, but no epithelium. Connective tissue near bone rather damaged by decalcifier, but intense nuclear infiltration and bone change marked as in A—more of it.

"O.im.1/12 not very satisfactory. Section thick and cellular changes not very distinct, but one gets the impression of much cellular infiltration. Rather an acute process involving bone.

"(e) Post ethmoid. Narrow long strip of bone. Nothing to add to A."

3.4118 (4 slides) April 6, 1921 (Case 32): "(a) Left middle turbinate. Not much increase of new or old connective tissue—in glandular areas very little, but here it is gorged with leucocytes and considerable serous effusion. Deeper there is fibrous hyperplasia and thickening of the periosteal layers and I should think the bone areas show an increase in thickness. There is apparently some cellular hyperactivity along the bone edges, but the definition is poor. *Old chronic inflammation with acute exacerbation.*

"(b) Another section shows rather marked subepithelial serous and leucocytic infiltration in another locality. Surface epithelium not much altered, otherwise as above.

"(c) Post ethmoid. Very much more new connective tissue formation and a good deal of leucocytic infiltration with numerous lymphocytes. Bone shows up badly under high power, but I should think it at least somewhat—probably much invaded, a subacute inflammatory process more plainly evident in the soft parts.

"(d) Sphenoid. Piece too small and bone unsatisfactory, but probably seat of inflammatory activity, i.e., ragged edges and chromatin effusion—cellular bone changes not shown.

Dr. Harrison L. Martland, pathologist of the Newark City Hospital, also examined my sections and dictated the following:

"Most sections show intact mucosa, in many places it seems to be edematous and hyperplastic; submucosa varies all the way from normal to areas containing numerous small mononuclears, practically no polymorphonuclears, some sections show considerable number of eosinophiles, indicating low grade chronic infection.

"Submucosa is often edematous, in some places there is questionable rarefying osteitis. Many sections show considerable dilatation of submucosal vessels (acute hyperemia) and undoubtedly represents low grade chronic inflammation, which is non-suppurative.

"*Conclusion:* It is quite possible in non-suppurative inflammation that edema of mucosa and submucosa with acute hyperemia of vessels, etc., may produce more pressure than a suppurative process, in which the pressure is often relieved by the breaking down of the tissues."

Case 30: Miss R. P., aged 25, was referred by Dr. Verhoeff at the Eye and Ear Infirmary on February 18, 1921, with diagnosis of bilateral retrobulbar neuritis. History of influenza two months previously, was in bed two weeks and had considerable trouble with her head. Always subject to colds accompanied by pain in the back of head and above eyes. There was, however, no pain through the eyes with the above-mentioned attack, but there was severe pain in the back of the head. The right eye was blurry almost with the onset of the influenza and two days later the left eye was likewise affected. As far as the patient can tell the vision of 20/200 has remained practically unchanged since the trouble commenced. The fundi were negative except small pigment spots in the macular lutea and a striped appearance, probably not pathological. Visual field showed slight contraction. The pupil of the right eye was somewhat larger than the left. A deflection of the septum to the right posteriorly, crowded an enlarged middle turbinate and evidently blocked the posterior sinuses. The left middle turbinate was also moderately hypertrophied. The physical, neurological, Wassermann and dental examinations were negative. Dr. H. H. Vail went over the case carefully, but found no evidence of any cerebral or cerebellar lesion. Dr. A. S. MacMillan made stereoscopic plates and reported that the ones underexposed showed slight haziness of the sphenoids. In the absence of other findings it was felt that the sinuses should be drained, so on February 24 a semi-radical sphenoid exenteration was performed on the right side. Smears and cultures were made from the sphenoid and numerous specimens saved for future study.

The walls of the sphenoid were pinkish and somewhat thickened and suggested a recent inflammatory condition. The patient was apparently not benefited by this operation. Three weeks later she was thoroughly reinvestigated and a lumbar puncture done, but the findings were practically negative. Dr. Alexander Quackenboss, who later examined the eyes, reported that there was a suggestion of an old iritis on the right, *i.e.*, a ring of deposit on the anterior capsule. He offered the suggestion that the loss of vision might be due to some focal infection, such as teeth or tonsils. The teeth appeared negative, both from films and on inspection, so the tonsils, which seemed somewhat enlarged, were removed, but no pus or special pathology was found. Following the lumbar puncture the patient was confined to bed for two or three weeks with very severe pain throughout the head. This pain entirely disappeared after the removal of the tonsils. Although the patient could not apparently read the chart better at the time of discharge than when she entered the infirmary, she could, when ordered to do so by Dr. Verhoeff, read ordinary book print, so he was inclined to think there was an element of hysteria in this case.

As to whether earlier drainage of the accessory sinuses would have proven beneficial, it is impossible to state. Cases of two months' duration, as was this one, do not respond as readily as those of short standing. These cases are followed for months or even years, so it is hoped that some future developments may throw light on the etiology. The cultures from the sphenoid showed one or two colonies of staphylococcus albus. For a report on the sections taken from the middle turbinate and sphenoid, see Dr. Wright's interpretation. A recent letter from the patient says that the vision remains the same, although the general health is much improved.

Case 31: Miss J. I. B., aged 27, was referred by Dr. Alexander Quackenboss on March 15, 1921, with diagnosis of optic neuritis, right. History of fair general health, but was rather tired. Had double pneumonia 4 years ago, since which period has been subject to colds and has had one for the past 4 or 5 days. This was accompanied with pain about the eye, which has increased in severity so that when seen she looked extremely ill. Eye was sensitive to light and on movements and pressure. Vision when first seen was 20/20. The right middle turbinate was somewhat enlarged and the septum bent slightly to that side. No secretion was seen within the nose, but there was a marked pharyngitis with considerable tenacious mucus. Transillumination was negative. The patient was referred to the Eye and Ear Infirmary for investigation and the sinuses

X-rayed by Dr. MacMillan, who reported: "Frontal sinuses undeveloped; ethmoidal region quite cellular; right slightly clouded, especially posterior group; sphenoids moderate size and well anterior to sella appear clear; antra are both large and clear." The physical, neurological and Wassermann examinations were all negative. Films of teeth showed infection about one which was extracted some 3 weeks later. On March 17, Dr. Vail examined the patient and found the vision 20/40 with central scotoma for colors. The following she was seen by Dr. Quackenboss, who found considerable increase in the neuritis and vision 10/100. There had been severe pain about the eye for the entire week and it was still sensitive to pressure. In view of the negative neurological examination, the rapidly diminishing sight and the increase in the inflammation of the optic nerve, it was deemed advisable to open the accessory sinuses. The patient was accordingly admitted to the infirmary, and on March 18, under general anesthesia, the right middle turbinate was removed, the sphenoid opened and the posterior ethmoid uncapped. The tissue was somewhat inflamed, but no pus was seen. The lining wall of the sphenoid was little, if at all, changed. Cultures and smears were made and specimens saved for study. On the day following the operation the patient felt considerably relieved and the eye was less blurry. This lasted but a few hours, then there was a rapid recurrence of the blurring and the vision continued to fail, so that a week after operation fingers could be made out only at 3 inches. Dr. Quackenboss examined the eyes 4 days after the operation and reported edges of disc practically obliterated, some exudate; small blood vessels engorged; no marked swelling or papilloedema; no hemorrhage. Two weeks after operation the swelling in the nose had subsided and the patient was much better; discomfort and blurriness alleviated; counted fingers at 8 feet; 4 days later fingers at 25 feet and vision was 20/80. On April 15, Dr. Quackenboss examined the fundi and reported blurriness practically disappeared and outline of disc sharply defined. April 25, the vision was 20/60. On April 29, Dr. Quackenboss again examined the eyes and noted slight pallor of the disc. The smear from the sphenoid showed only blood and a few epithelial and pus cells. In one of the culture tubes there were 3 colonies of diphtheriod bacilli. The specimens from the middle turbinate and sinuses were examined by Dr. Wright. See his report.

Case 32: Mrs. H. G., aged 24, referred by Dr. H. B. C. Riemer at the Infirmary on March 16, 1921, with diagnosis of bilateral retrobulbar neuritis. History of frequent headaches, otherwise negative.

Present illness: Following a severe cold 4 weeks ago there was a sudden loss of vision in the left eye and 2 days later in the right. This was associated with pain on moving the eye and sensitiveness to pressure. When seen a week or ten days ago by Dr. Riemer, there was considerable swelling about both discs and the vision was practically the same as on admission, *i.e.*, 20/70 right and fingers at 2 feet left. Fundi examination showed that the edges of the discs were not entirely lost or obliterated. The lower outer edges were quite distinctly made out. Upper portions indistinct with some pallor. Slight enlargement of the fine capillaries. Physical, neurological, dental, Wassermann and spinal fluid examinations negative. The septum was badly deflected high up with enlarged middle turbinates in close contact with it. Pus above the right middle turbinate and below the left. Dr. MacMillan reported as to the X-rays: "Both frontals small and clear; left ethmoids definitely clouded, right ethmoids clear. Both antra clouded, probably thickened membrane. Teeth show no evidence of apical involvement. Teeth in good condition." Dr. Vail went over the patient neurologically and found no evidence of any cerebral or cerebellar lesions. As the eyes had not improved during the past week and there was a slight pallor noticeable, it seemed urgent that the infection should be relieved, so on March 23, under general anesthesia, a submucous resection of the septum and a bilateral semi-radical sphenoid operation were performed. The sphenoidal mucosa appeared practically normal. Smears and cultures were taken and specimens sent to the laboratory for study. The smears were chiefly blood. Five cultures from the sphenoid showed 1 to 3 colonies each of staphylococcus albus. No pus was seen during the operation. Patient improved rapidly, so that when discharged a week after the operation the vision was 20/70 left and 20/40 right. On April 29, the fundi were examined and the edges of the discs were found to be sharply defined, but there was slight pallor of the entire discs. Vision without correction 20/30 both. See Dr. Wright's report on specimens removed from turbinates and sinuses.

Case 33: N. C., aged 24, was referred by Dr. Verhoeff at the infirmary on April 29, 1921, with acute unilateral retrobulbar neuritis. Patient in good general health. Has had several operations on nose and throat. Tonsils were removed last November and septum resected(?) last January. Has had considerable secretion in throat and blowing of nose. No recent cold. On April 10, on awaking he noticed that things seemed blurry when viewed by left

eye. This grew rapidly worse so that in 2 days he did not even have light perception. Four days later the vision returned somewhat so that he could count fingers at 5 or 6 feet, but since that time it has been stationary. Dr. Verhoeff, on examination, noticed distinct pallor of the left disc. Eye sensitive when moved up or sideways, and there is a sense of discomfort back of eye. There was a moderately high posterior deviation of the septum to the left, which was in contact with a hypertrophied middle turbinate. There appeared to be considerable obstruction to the drainage of the posterior sinuses on the left. No secretion was seen. Transillumination negative. Naso-pharynx showed marked reddening with thickened glandular areas scattered over it. Medical, neurological and Wassermann examinations negative. Visual fields showed a marked central scotoma and some contraction. Massachusetts General Hospital reported that there was no other cranial nerve involvement. Dr. Vail ruled out cerebral and cerebellar lesions. Dr. MacMillan reported on X-ray of the sinuses as follows: "Posterior ethmoids both sides are definitely clouded, sphenoid does not appear to be quite normal. Antra and frontals do not appear to be affected. Teeth: Abscess about roots of right lower second molar." On May 3, the patient was operated upon. It was found that the upper posterior portion of the septum which appeared twisted, had not been resected. This was accordingly done by Dr. Vail. The resection extended back to the front wall of the sphenoid, as it was otherwise difficult to obtain a good view of that region. The middle turbinate was removed. The front wall of the sphenoid broken down and the posterior ethmoid cell uncapped. Various sections of the sphenoid and posterior ethmoid were preserved for study. Smears and cultures were made, one on liquid blood serum in an endeavor to grow the streptococcus viridans. The lining membrane of the sphenoid appeared normal. No pus was seen. Within 48 hours the discomfort about the eye and sensitiveness to pressure had entirely disappeared. Fogginess less dense. Not very marked reaction from operation. Vision fingers at 12 feet. Two days later patient had a temperature of 103° and the lateral wall of the throat showed some redness. This was followed late in the afternoon by a severe hemorrhage from the left side of the nose, which was not controlled until the posterior nares and nose were firmly packed for 24 hours. Two days later the patient developed acute middle ears and it was necessary to incise both drum membranes. The slight infection in the nose subsided rapidly and although it was necessary to reincise the drum membranes, the patient went along favorably.

Cultures from the sphenoid showed 2 colonies of staphylococcus albus on solid media. A few short chains of streptococcus in water of condensation in addition to abundant staphylococcus in blood serum. The patient is still under observation. On May 31, 1921, fingers could be made out at 20 feet.

SUMMARY.

Etiology: Earlier writers considered the anatomical relations of the optic nerve to the accessory sinuses the chief factor. Later ones claimed the trouble was transmitted by the soft tissue. No one etiological condition responsible for all cases. Purulent infection accounts for a few, while hyperplasia seems of minor importance. The size and position of the middle and superior turbinates probably explain the etiology in a large number. Poor ventilation and faulty drainage are all important predisposing factors.

Pathology: Subdivided into four parts: 1: Direct extension; 2: Toxemia; 3: Bacteremia; 4: Hyperplasia.

1: By direct extension acute infections in the adjacent sinuses spread by continuity of structure to the sheath of the nerve, or even to the optic nerve itself, while in the more remote sinuses the infection becomes walled off. Nature has also walled off the chronic infections so that it is futile to longer believe purulent sinus disease the one and only etiological factor. Cases with sudden loss of vision presenting such noses might better arouse suspicion that the cause should be sought elsewhere than longer be considered the *sine qua non* of accessory sinus blindness.

Similarity between optic neuritis and Bell's-palsy.

2: Toxemia. Toxins originating in the accessory sinuses or elsewhere may involve the optic nerve.

3: Bacteremia or focal infection. Micro-organisms may be carried in the blood stream or lymph channels from the foci of infection in the sinuses to the regions adjacent to the optic nerve. Hematogenous focal infection of the nervous apparatus has been demonstrated, so why not a similar involvement of the optic nerve?

4: Hyperplasia. Probably as a predisposing factor of considerable importrance, but not per se. Hyperplasia plus infection and direct extension to the optic nerve responsible for many cases.

Cultures when taken all gave some growth, principally the staphylococcus albus. A few streptococci were found, but the viridans were not isolated. Several of the sections showed considerable increase in the leukocytes and fibroblastic tissue along the margins of the bone. Others showed chronic inflammation of the deeper ele-

ments of the mucosa and of the bone, while a few showed fibrous hyperplasia.

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ANTROSCOPY OF THE MAXILLARY SINUS.*

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Those who have followed the recent literature in rhino-laryngology have no doubt noticed the divergence of opinion there exists among authorities, on the value of our present available aids in the diagnosis of disease of the Antrum of Highmore, and the method or methods of procedure in the treatment of this condition.

Special attention is called by many to the unreliability of the X-ray, transillumination and clinical findings as an aid in the diagnosis of maxillary sinusitis. Some even call the X-ray findings misleading, having observed negative results on antrum puncture where pus was expected according to the X-ray interpretation; and again finding definite pathologic lesions and even suppuration where X-ray examination apparently gave negative results.

Transillumination seems to have lost favor with others.

As to the value of clinical signs and symptoms it is claimed that one should put little dependence to them, as changes in the turbinates are not always present, since the disease may be confined to the interior of the antrum only. This situation especially confronts us in the diagnosis of early cases. Nasal discharge may be absent or removed by the patient before being examined.

Opinion varies regarding therapy from the very conservative long drawn period of irrigating the antrum, to very radical operative methods. Most surgeons however, agree on the necessity of radical exploration of every suspected antrum.

It is not my object in this preliminary report to agree or disagree with any of the views cited above, but rather to point out a method by which a definite diagnosis may be made, before any treatment is advised or undertaken.

It has occured to me that were it possible to inspect the interior of the antrum by direct vision, and so obtain a good view of its contents and the condition of its lining membrane, one could easily establish the presence or absence of a pathologic process, and thus in a rather quick and simple manner override all the difficulties and speculations associated with the diagnosis of the presence or absence of a diseased Antrum of Highmore.

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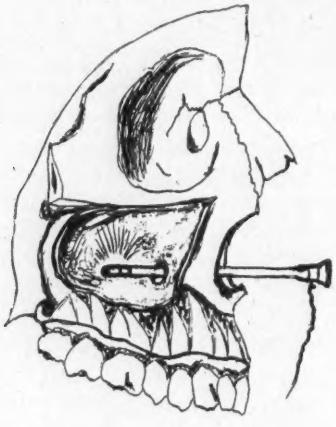
All that is necessary for the procedure is (1) a thin calibred instrument by means of which a direct view of the interior of the antrum could be obtained, and (2) a small opening in the antrum to admit this instrument.

The first was easily found in the Holmes naso-pharyngoscope, and the second accomplished with a straight antrum trochar.

Under local anesthesia (cocaine adrenalin packing) the antrum was punctured in the usual manner, special pains being taken to make the puncture as far anteriorly and towards the floor of the nose as possible, so as to permit inspection of the greater part of the antrum. After clearing the field of blood and mucus, the naso-pharyngoscope was introduced into the antrum and the interior inspected.



After overcoming minor difficulties (chiefly that of keeping the fenestra and bulb free from blood and secretions) a good view of the contents and lining membrane of the antrum was obtained.

The normal state of the lining membrane was observed and found to be similar in character to that in the nasal fossae. Pathologic processes such as congestion, hypertrophy, atrophy and polypoid degeneration of the membrane could be observed. Of special interest was the similarity in appearance of polypi found in the antrum to polypi arising from the ethmoids or turbinates. In cases where pus was absent, the cavity was frequently found filled with a small quantity of a thin gelatinous fluid. The latter was found more plentiful when polypoid degeneration was present.

By such direct study of the gross pathologic lesion present, no difficulty at all was found in deciding upon the correct diagnosis and the method of treatment to be employed in each instance.

The following diagram is self explanatory.

The following is a preliminary report of four cases where antroscopy was used to establish the diagnosis. The comparative value of other aids in the diagnosis of maxillary sinusitis, and methods of treatment is carefully considered in a paper now under preparation.

Case 1: B. S. female 23 years. For two years marked nasal discharge. Headache and feeling of discomfort in left side of face. Left upper bicuspids extracted due to root abscess of both. X-ray showed cloudy left antrum. Antroscopy revealed polypoid degeneration of the membrane lining the facial, orbital and surfaces zygomatic of the antrum. Patient was advised a radical operation and referred to the hospital.

Case 2: S. G., female 18 years. Frequent colds and nasal discharge. Pain in the left side of the face. X-ray showed a slight cloudying of the left maxillary sinus. Antroscopy showed a large single polyp on the facial surface and a small polyp on the orbital surface. Patient is under observation.

Case 3: M. C., male, 26 years. Frequent attacks of rhinitis and sneezing. X-ray examination showed a cloudy left antrum. Antroscopy revealed marked congestion and thickened mucous membrane. No polypoid changes visible. Patient is treated by irrigation and progress observed by antroscopy.

Case 4: L. H., male, 17 years. Running nose for over two years. Soils several handkerchiefs daily with a very thin mucoid secretion. X-ray showed both antra cloudy, mostly left. Antrum puncture and washing gave negative result. Antroscopy revealed marked congestion throughout the antrum and areas of thickened membrane. No degenerative changes noticed in either sinus. Marked and immediate improvement followed irrigation of the antra. Patient is still under treatment.

211 Henry Street.

CALCULI OF THE NOSE AND THROAT—REPORT OF CASES.

Dr. Lee W. Smith, Butte, Montana.

I beg to submit the following report, two cases of calculi, which have come under my observation recently.

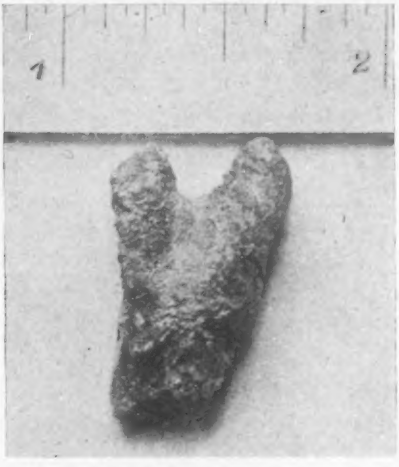
Case No. 1: P. R., age 28, single, white: occupation, miner. Came to the office October, 1920, complaining of pain in the right side of throat and neck—difficulty in swallowing, and a sensation of fullness in the throat. Family history negative. Past history: Has been a miner for about four years—works underground the majority of time. Had measles and mumps in childhood. Patient states that he has had considerable trouble with throat, an occasional tonsillitis, and at one time had to have throat lanced.

Examination shows a fairly well developed male. On looking into the throat near the center of the anterior pillar, a small round wound was seen which looked like an opening into a sinus, most probably, I thought, that of a peri-tonsillar abscess, which had broken through. However, on probing this, a hard mass and a grating sensation was felt. With a curved probe and by enlarging the wound with a small incision in the anterior pillar a calculus (Figures 1 and 2) was extracted without any difficulty. Patient immediately experienced sense of relief. Was told to report the following day for observation but failed to return.

Case No 2: R. P., age 65, married; occupation, cement worker. Patient was referred for examination of nose and throat. Complaint—difficulty in breathing. He had been to see his family doctor

for treatment of chronic eczema, and casually mentioned that he could not breathe very well through his nose.

Family history negative. Past history: Has always been in good health with the exception of the last six months, during which time has been troubled with an eczema of hands and wrists. Patient has worked as a cement worker for the past thirty years, and states



that he has often had to blow small particles of cement from the nose. About four months ago he first noticed a hard mass in right nostril which he tried to remove but was unable to do so, and since that time has had a gradually increasing difficulty in breathing through the nose.

Examination shows a well developed elderly male with an eczema on wrists and hands. His family physician reports entirely negative physicial findings, together with negative laboratory reports of

blood wasserman and urine. Nasal examination shows a perforation of septum with a foreign body (Figure 3) threaded through it. From the picture of this foreign body it will be seen that there are two horns with a groove in the middle. The groove was riding on the edge of the perforation, anteriorly, and the two side enlargements or horns, prevented its removal or falling out. On gentle manipulation, the posterior end of the foreign body was pried outward into the right nostril, and pushed upward, then removed in toto through the right nares. The perforation through the septum was about 1½ cm. in diameter.

Resume: The first case is interesting from the standpoint of size of calculus with so few symptoms resulting from it. Calculi of the tonsillar cryps are relatively common but usually are much smaller. This calculus was apparently situated between the tonsil and anterior pillar, and there is a possibility that it may have started at the time he had the peri-tonsillar abscess several years ago.

The second case is probably a concrete calculus, due to the deposition of concrete dust within the nasal perforation and increased to its present size, by the slow addition of dust "setting" itself by virtue of the moisture of the nasal secretions.

Silver Bow Club Building.

LARYNGECTOMY FOR CANCER OF LARYNX WITH MODIFIED TECHNIQUE AND ATTEMPTED FORMATION OF SKIN GRAFT TUBE IN PLACE OF LARYNX.

DR. R. H. GOLDTHWAITE, Camp Benning, Ga.

Complete laryngectomy for cancer of larynx was first performed by Billroth, but early operations were attended by enormous mortality and the operation fell rather into disrepute. Of recent years the technique has been developed to the point that the operation may be approached with reasonable confidence. Gluck's method is to start from above, separate the larynx from the pharynx and then work down, cutting the trachea last. Keen has reversed the procedure, and cuts the trachea first and then works up to the pharnyx. Some operators prefer a preliminary tracheotomy, others do a one-stage operation. There are advantages and disadvantages to it, but if the trachea is to be

brought out to the skin and fastened there, the one-stage operation is preferable. The article of MacKenty in the *Journal A. M. A.*, September 15, 1917, is full of useful suggestions. The feature that holds most terror for the patient is his loss of voice, and so far all attempts in this direction have been in the way of supplying an artificial larynx. Braun's and Kluck's are the best known of these. The fact that if a column of air under pressure can be introduced in the pharynx, articulate speech can be formed, suggested that if a continuous skin tube could be formed from the pharynx to the trachea stump then speech would be possible without cumbersome apparatus. If the tumor growth did not involve the epiglottis, it seemed probable that leakage could be prevented after healing occurred and it was planned to avoid aspiration of septic material from the throat and necrosed skin from the graft, by temporarily bringing the tube out over the clavicle until complete skin formation had occurred and the opening into the pharynx had healed. Then by second operation the newly-formed tube could be brought in line with the trachea.

Mr. G. W. K., age 65; occupation civil engineer.

Family History: Father died in 1886 with bowel trouble. Mother died at 80 years of age. No cancer in the family. Previous history mastoiditis operation complicated by lateral sinus thrombosis and operative resection of jugular vein, left side.

He first noticed hoarseness in November, 1917. This continued and he consulted a physician in January, 1918. No change for several months. In May, 1918, he was seen by Dr. Eagleton, of Newark, who recommended removal of growth on the vocal cords. This was done in June, 1918, by the laryno-fissure method. The growth was excised and tracheotomy tube installed for 24 hours. Convalescence lasted four weeks, during which time breathing was partially through the wound and partially through the mouth. Voice was not recovered, but he was able to make himself understood by a hoarse whisper. He was seen at intervals by Dr. Eagleton until November, 1918, and by Dr. Baker until August, 1919. Weight during all this time was keeping at normal level of 158. It was only in January, 1920, that loss of weight began and became progressive so that by May he was definitely loosing about three pounds a week.

In April, 1920, he was first seen by me. Larynx at this time presented an irregular mass with an irregular stenosed passage which seemed barely sufficient for breathing purposes. No ulcerations at this time. In view of the laboratory report from the specimen removed at the first operation, "chronic inflammatory tissue,"

no intervention was advised and the case was observed at weekly intervals for a couple of months. During this time the progressive loss of weight and increase in size of the growth became so definite that the question of operation was taken up. From the size and extent of the growth, with the fact of recurrence after laryngo-fissure operation of two years previously, it was felt that nothing short of laryngectomy would be effective. He was seen again June 26, 1920, by Dr. Eagleton, of Newark, who concurred in this opinion.

June 30, 1920, a tracheotomy under local anesthesia was performed and a ten-day period allowed before further operation so that his lungs and trachea might become thoroughly accustomed to the new air conditions. The improved oxygenation obtained by the resulting easy and ample supply of air was a great benefit, and after the second day he was up and about. July 12, the operation of laryngectomy was performed under chloroform oxygen anesthesia by the intratracheal route through a soft rubber catheter introduced inside the tracheotomy tube. The presence of the tube healed in position made this simple and non-irritating and a large sheet of rubber dam was set around the tube, much as the dentist isolates a tooth he is about to fill with gold. The upper part of this apron was laid back over a wire support so that free flow of air went on beneath it, isolating the breathing and anesthesia from the operator until in the course of the operation he should have to cut the upper end of the trachea. This absolutely prevented the leakage of blood into the trachea during the operation. Anesthesia was skillfully administered by Captain Long and after the patient was under, a mild flow of oxygen was maintained only occasionally adding a little chloroform vapor as the patient showed signs of rousing. The patient was kept light by this means and was conscious soon after the operation.

A median line incision was made about five inches long and then dissection carried down to the thyro-hyoid membrane and the thyroid cartlidge. Whenever possible the perichondrium was elevated by a Freer elevator and lateral dissection kept close to the cartlidge. The omo-hyoid and thyro-hyoid muscles were severed to obtain more room and dissection carried well back on each side. The growth was found to have eroded the cartlidge in two places, but there had apparently been no extension into the muscle layers. This made a close dissection possible, and hemorrhage was kept at a minimum. Tissue ooze was controlled by working alternately on each side, and putting large gauze packs under the muscle layers on the side not being worked on. Everything was then dry at the

time it became necessary to cut off the trachea just below the cricoid. A gauze pack was immediately packed in around the top of the trachectomy tube to again seal off the trachea. Dissection then followed up the posterior surface guarding carefully the esophagus; then the thyro-hyoid and ary-epiglotidean folds cut, and the growth removed. Laboratory report (Maj. Sinclair) shows specimen to be squamous-cell carcinoma, apparently entirely removed.

The patient's condition was excellent and, as the removal of the epiglottis had not been necessary, it was decided to attempt the skin graft. A large size rubber tube of double thickness about four inches long was then coated with Thiersch skin graft from the non-hairy parts of the right upper and lower arm, and then introduced into the wound in place of the larynx. Four catgut stitches at the upper end attached the rubber tube to the cut ends of pharyngeal opening, applying the skin graft to the edge of the mucus membrane. A counter opening was then made through the platysma and skin over the clavicle about two inches to the right of the tracheal opening and the other end of the rubber tube with its graft brought out through it and fixed in position by transfixing the tube with a large safety pin.

Considerable discharge came out the counter opening in the neck for the next few days, but this discharge was easily kept away from the tracheal opening. July 16 the stitches at the upper end of the tube had loosened and the large tube was removed. After cleaning the wound, a smaller size rubber tube was replaced and thereafter removed and replaced daily. Murphy drip was given all night after the operation with a soda-bicarb, glucose and salt solution, then next day a schedule started whereby he received four nutrient enemas consisting of two eggs, one ounce of whisky and four ounces peptonized milk. Each feeding was followed by a free interval of three hours then Murphy drip was given for two hours. This method succeeded in keeping him sufficiently nourished and supplied with fluids until July 20 when feeding by stomach tube was started and the quantity of feeding rapidly raised to one pint; then July 26 he started drinking water through a glass tube. At first small amounts occasionally came down the false passage, but they ran out on the chest and did no harm. As soon as water was taken without leakage all liquid nourishment was given through the tube. August 2 he was examined with a bronchoscope and the fact of proper connection of the grafted skin and mucus membrane at the pharyngeal orifice definitely established.

August 10, operation under local anesthesia to connect the skin graft tube to the trachea. The dissection of the tube was not as difficult as had been expected and when the tube was cut through at the point where it dipped through the platysma, it was evident from inspection of its interior that a complete skin tube had been formed. The lower portion of the tube was rolled laterally towards the median line and the upper end of the trachea cleared by dissection, then the two tubes were drawn together by chromic gut, and fair approximation obtained leaving a space in front for the tracheotomy tube. A rubber tube with a gutter cut half way up in its front side was slid up into the skin graft tube and down into the trachea; then the tracheotomy tube introduced so as to fit into the gutter, and a suture tied around them both to hold them firm. Following the operation both these tubes were changed daily and cavity cleaned. For a few days following the operation feeding through stomach tubes was necessary on account of irritation to the esophagus at the area of the operation. This spasmodic contraction gradually disappeared and he was swallowing liquids by August 30 and September 10 was taking all soft solids. When the rubber tube is removed he can send a column of air up through the graft tube and talk in a hoarse hollow tone which was however quite intelligible across the room. The rubber tube was removed entirely September 15 and the skin tube became rapidly smaller from the progressive closing in of the scar tissue of the neck until it was reduced to a tiny passage, the general condition of the patient continued to improve and by September 27 he weighed 132 pounds, a gain of 22 pounds since the operation.

As it was impossible for him to pass air up around the tracheotomy tube into the skin graft tube, a small hole was cut in the tracheotomy tube about one-half inch back from the shank, and he could then send a column of air up through the skin graft tube and produce articulate speech, by simply putting his finger over the opening of the tracheotomy tube during expiration. Further contraction of scar tissue in the neck completely obliterated the skin tube during the next ten days. August, 1921, 14 months after operation, the patient is in the best of health, weighs 140 pounds and has no evidence of recurrence. This case demonstrates the possibility of making a skin graft tube in the neck. The difficulty yet to be overcome is to find some means to avoid the scar contraction which gradually obliterated it.

SOME REMARKS ON THE WEBER, SCHWABACH AND RINNE TESTS.*

DR. G. W. MACKENZIE, Philadelphia, Pa.

The subject of this paper was suggested by reason of the following facts:—

1. That these tests when carefully conducted afford the otologist the best qualitative means available for determining the anatomical site of the lesion responsible for loss of the hearing function, partial or complete.

2. Besides in the hands of a skillful examiner they may, when carefully timed in combination with air conduction tests with tuning forks, serve as quantitative tests subject to limitations as are other present day methods.

3. That in spite of the above mentioned facts there exists too often a looseness in the method of conducting the tests even by credited otologists. One may observe otological clinics here and there where an attempt is seldom made to conduct these few simple tests even in those cases undergoing treatment for deafness. The explanation for this neglect may be one of several.

(a) Forks may not be available, or what are available may be unsuitable for the purpose.

(b) Lack of faith in the tests brought about by a carelessness in technique which naturally leads to irregularity of findings and in turn to lack of interest. The same holds true in other fields. In photography, laboratory work or vestibular diagnosis, in fact in anything of a technical nature, careful technique is the one big essential to success.

(c) Lack of time to apply the tests.

(d) Lack of ability to conduct them.

(e) Indifference.

No ophthalmologist can hope to practice his specialty successfully without making careful tests for visual acuity, visual fields and color perception, neither can an otologist hope to practice his specialty successfully and neglect the subjective hearing tests that call for more time and skill than is required of the ophthalmologists in the making of his special tests.

As pointed out earlier these tests are the best qualitative means available for determining the site of the lesion responsible for loss of hearing. To be sure with the otoscope we are able to inspect the drum membrane and determine the presence or absence of re-

*Read befoer the American Academy of Ophthalmology and Oto-Laryngology, Philadelphia. Oct. 19, 1921.

which when present tell us that the middle ear is involved; but which when present tells us that the middle ear is involved; but from the otoscopic appearance alone we could not say positively that the middle ear condition is responsible wholly or in part for the impairment of hearing in a given case. The history may afford some assistance but it is not always to be depended upon for it may be found by the functional hearing tests that the hearing is impaired by reason of a lesion in the perceiving apparatus, a neurolabyrinthitis. For instance, a physician friend presented himself for treatment of bilateral impairment of hearing, due according to his opinion to a middle ear catarrh, since he had observed that with every cold in the head the hearing would decrease perceptibly and after the cold subsided the hearing would improve; however with this last cold the impairment was more pronounced than usual and was lasting longer, a history suggestive of middle ear trouble. The otoscopic examination revealed a somewhat dull, retracted, opaque membrane that was unduly flaccid on both sides. These findings together with the history would seem to clinch the diagnosis of bilateral middle ear catarrh. On the other hand the functional hearing tests revealed the Weber lateralized to the better hearing side, the Schwabach shortened on both sides. Rinne positive and the air conduction shortened. These findings did not match up with the history and otoscopic findings of middle ear trouble but were characteristic of disease of the perceiving apparatus.

Which of these evidences was one to believe, the history and otoscopic appearances on the one hand or the functional hearing tests on the other?

I accepted the functional hearing test findings without hesitation and asked the doctor what medicine he had been in the habit of taking for his colds in the head, and he answered by saying that he took a compound tablet known as Cinchonin salicylate in appreciable doses. This corroborated the functional hearing test findings, and accordingly the medicine was discontinued and nux vomica substituted for antidotal purposes with complete success. This is not a rare case. We all meet them now and then; occasionally they are recognized but more often they are not.

Experience eventually teaches us not to rely absolutely upon a patient's history, for in the case just cited the history volunteered by the patient was incomplete. The more complete history was obtainable only after the functional tests suggested further inquiry. Experience teaches us not to rely absolutely upon otoscopic appearances. When it comes to estimating the amount of hearing function we frequently find the worse appearing ear otoscopically to be the

better hearing one. It is not a rare occurrence to find a membrane appreciably retracted with good hearing, in some cases approaching normal. On the other hand the functional hearing tests, carefully conducted, cannot deceive us. They can however if loosely conducted. Wherefore the plea for careful technique is emphasized.

After having obtained accurate and well balanced findings it is essential that the otologist possess a sufficiently wide knowledge of the subject of otology in order to properly interpret them.

In a little more than half the number of cases we are called upon to treat, the findings will be found to correspond typically to a disease of the conducting apparatus pure and simple, unilateral or bilateral, or to a disease of the perceiving apparatus pure and simple, unilateral or bilateral. In somewhat less than half the number the findings will not correspond typically to one of these conditions but will suggest a mixed condition, unilateral or bilateral or even a disease of the conducting apparatus of one side combined with disease of the perceiving apparatus on the other. In fact the variety of combinations are legion. In all of them it is essential to successful treatment that the examiner should be able to determine just how much one part is involved and how much the other. Furthermore whether the involvement of the one part is primary or secondary to the other. Finally if they are not associated conditions which of the two antedated the other.

The vestibular tests, important though they are, have so fascinated otologists in recent years as to divert attention from the functional hearing tests, which are vastly more important as measured by the comparative frequency of the conditions calling for these separate forms of tests. In other words there are scores of cases of deafness to every one of vertigo. No matter whether the problem is one calling for vestibular tests or one calling for functional hearing tests or both combined, the more loosely they are conducted the more irregular will the findings be and the more irregular the findings the less dependence will be placed in them, all of which eventually leads to a distaste for if not an actual condemnation of the tests.

A few years ago a low ebb was reached as to the estimated value of the vestibular tests, all because of irregularity in the findings resulting from faulty technique, which in turn suggested immoderate speculations as to the reason rather than a closer inquiry into the technique employed, the lack of which was fundamentally responsible for the irregularities.

Concerning the technique to be recommended in the making of the functional hearing tests the writer will present only those which

he feels to be most essential to success, granting at the same time the possibility of improvements.

Of first importance is the selection of a suitable fork. Ordinarily the three tests (Weber, Schwabach and Rinne) are conducted with the C fork of 256 double vibrations, weighted at the ends to suppress overtones. The prevailing type seems to be the one adopted by Politzer and made by Reiner. This is the fork the writer began his work with and still uses. It has many advantages and a few disadvantages. During the last fifteen years the writer has been fortunate enough to have had several forks of the same design made by other manufacturers donated to him. In trying them out it was found that they showed very wide discrepancies in the duration of vibrations (variance in decrement.) The shortest audibly vibrating fork of this model was 56 seconds. This fork gradually lessened until it eventually ceased to vibrate altogether because an imperceptible crack in the beginning had widened eventually to a wide split. The second shortest was 70 seconds. The longest vibrating fork was 250 seconds. With the fork of 70 seconds vibration the positive Rinne with normal individuals was ascertained to be about 20 seconds. With the 250 seconds fork the Rinne was positive in the same individuals about 150 seconds. With the 20 seconds positive fork it was found that the normally positive Rinne findings were considerably more irregular and inconsistent than with the 110 seconds vibrating fork of Reiner make. With the 250 seconds vibrating fork of an American make the fatigue element entered so largely, in spite of efforts to avoid it, that the findings were not at all reliable. Furthermore several forks of the same model made by the one manufacturer were found to vary considerably in their rate of decrement. After considerable experimenting it was found that the ideal fork was one that vibrated somewhere between 100 and 120 seconds. The chance for error increases in proportion as the figures increased above 120 seconds or decreased below 90 seconds. The longer duration forks, besides increasing the chances of fatigue of the patients auditory apparatus and the examiners fingers, consumes an excessive amount of time in making the tests, especially the Rinne. Furthermore it is more difficult for the patient to tell exactly the moment he ceases to hear the fork in the case where the decrement is so gradual than where the decrement is more abrupt as occurs in the case of the shorter duration fork. On the other hand with a fork of relatively short duration the rate of decrement is so abrupt as to increase the difficulties in estimating the finer grades of positive and negative Rinne that are possible with the longer

vibrating forks. In short the best all round fork is one that vibrates neither too long, more than 120 seconds, nor too short, less than 90 seconds.

In order to obtain an ideal fork a fork maker was instructed to manufacture a fork of 256 DV weighted at the ends that would vibrate approximately 110 seconds to take the place of my Politzer-Reiner fork in the event that something might happen to it. He followed the instructions given and presented a fork that vibrated 256 DV with weights whereas the Politzer fork was 256 DV without weights and decidedly lower with weights.

With the new fork of exactly 256 DV with weights it was found that although the total audibility by air conduction at three-fourths inch from meatus was 110 seconds, the same as that of my Politzer fork, generally considered to be 256 DV but actually 160 DV, there was a very wide difference in the Rinne findings in normal individuals. The normally positive Rinne with the Politzer fork is 40 seconds whereas the normally positive Rinne with the Standard fork was found to be about 75 seconds.

After considerable experimenting with the new fork it was abandoned in favor of the old Politzer. Accordingly the instrument maker was directed to make another fork with the object of eventually standardizing it after my Politzer fork, which is 160 DV with the weights and not 256 DV as many supposed it to be. At the same time other improvements have been added.

It is well that in the constructing of a tuning fork attention should be given to the handle. The Reiner fork has a relatively thin, at the same time, smooth handle. The thin handle tends to cramp the fingers more than a thicker handle. A smooth metal handle tends to become slippery after a while, especially if the hand of the examiner is inclined to sweat. A thicker handle of gutta-percha is preferable. If the tip of the handle is small or sharp at the circumferance or pitted at the center it will be less comfortably tolerated on the patient's mastoid than if free of these defects.

Having satisfied oneself that he has a fork well adapted to the purpose he is ready to begin testing.

The Weber Test: The intention of the Weber test is to determine whether the sound of the vibrating fork is heard better on one side than on the other or equally well on the two sides when the tip of the handle is pressed moderately firm against the skull just below the hair line, midway between the two ears. The fork should never be applied to the teeth since it is unsanitary to say the least, besides in the presence of a maxillary sinusitis of one side the secretion contained in the cavity together with the thick-

ened mucous membrane will tend to impair the conductivity on that side. In the presence of normal hearing the patient does not lateralize the sound. In the case of unilateral disease of the conductive apparatus the sound is referred to the diseased side, in the case of bilateral disease of the conductive apparatus the sound is referred to the worse hearing ear. In the case of unilateral disease of the perceiving apparatus the sound is referred to the normal hearing ear and in bilateral disease of the perceiving apparatus toward the better hearing ear. In the case of disease of the conducting apparatus of one side and perceiving apparatus of the other it will be referred to the side with the conducting apparatus disease. In the case of bilateral mixed condition (middle ear disease with secondary internal ear involvement) it will be referred to the side with greater amount of middle ear involvement or the side manifesting the less amount of inner ear affection.

At the present stage of otologic development it is hardly sufficient to determine merely that the Weber in a given case is lateralized to the right or left. For in one case where the record reads "Weber lateralized to the right" the lateralization may be so slight as to be almost indifferent, whereas in another it may be lateralized so strongly as to be referred to the right side even when the fork is placed over the left mastoid. There is a considerable difference in the overbalancing of the Weber in these two cases and also in the meaning. Therefore it would be better to determine just how strongly the Weber is lateralized and signify it by an adjective such as mildly, moderately strong or strongly to the right. It would be better still if we designated it approximately in inches. For instance, if the sound of the vibrating fork is lateralized, let us say to the right, when placed in the middle line of the skull it should be gradually brought past the middle line to the left until a point is reached where the patient feels doubtful about the lateralization or begins to lateralize the sound to the left. If it is two inches or four inches let it be so recorded in the findings.

The writer has been in the habit also of timing the Weber, not because it is absolutely necessary but to serve as a check on the Schwabach test, when that test is reached. If a discrepancy is found to exist between the Weber and the Schwabach it tells us that there has been an error somewhere; in which case a repetition of tests becomes necessary until the error is discovered and corrected.

Schwabach Test: This test calls for even more care than the Weber. The same fork is applicable as was used in the former

test. The Schwabach test is commonly made too low down on the mastoid; the writer's preference is for the hollow area corresponding to the antrum region behind the pinna, for the reason that the anatomical conditions at this point tend to correspond more nearly on the two sides than they do elsewhere on the mastoid.

The object of the Schwabach test is to determine whether the patient's bone conduction is better or worse than normal. This can be more accurately determined when it is timed with a stop watch than when it is merely guessed at. One must be sure that the control is normal, which can only be ascertained after making repeated comparison tests with other normals. If the examiner is not normal he must be careful to select an individual who is normal for use as a control. I have known examiners who were not normal themselves attempting to make corrections or allowances for their error but never with any degree of satisfaction. Occasions however arise where correcting allowances must be made. It happens in those cases where there is a marked difference in the ages of the examiner and the patient. It is common knowledge that there is a perceptible difference in the hearing by bone conduction in children and old people in favor of the former.

When making the Schwabach test the examiner should be careful to apply the fork to corresponding areas over the mastoid of the patient and the control and with equal pressure, for the more firmly the fork is pressed on the mastoid the better and longer it will be heard. Attention should also be given to the possibility of fatigue of the hearing organ. For instance if a vibrating fork is applied to the mastoid of any one, normal or otherwise, and is steadily held there for 20 seconds or more or until it is no longer heard and then removed for 5 or 6 seconds and reapplied to the mastoid it will again be heard. During the 20 seconds or more it was first applied to the mastoid the hearing organ became fatigued when the fine sound could no longer be heard. During the 5 or 6 seconds pause recuperation occurred sufficiently to permit of hearing again an even less intensive sound than was possible when the organ was fatigued. To lessen the chances of fatigue it is well to avoid applying the fork when it is vibrating too loudly. By the time this test is reached one can generally estimate about how long the patient will probably hear the fork on the mastoid and begin applying it 20 or 30 seconds before it is expected the hearing will cease. At the same time attention should be paid to rests. The best method is to interrupt its application every 2 seconds, that is to say, apply the fork for 2 seconds withdraw it for a like period thus alternating until the patient is sure that he

hears it no longer. Again this alternating interruption allows the patient a better opportunity to determine the difference between feeling and hearing the vibration, which at times is quite difficult with many patients when the fork is applied continuously.

It is generally known that when the bone conduction is better (longer) than normal it speaks for a disease of the conducting apparatus and when it is less (shorter) than normal it speaks for a disease of the perceiving apparatus. But this is not absolute for there are cases where the bone conduction may be less than normal on one side in the presence of normal air conduction because some of the bone conduction on the normal side is loaned, so to speak, to the opposite side as occurs in the case of a pronounced nerve deafness. Again there are cases where the bone conduction is better than normal in the presence of normal air conduction, because it had more than a normal amount of bone conduction to borrow from the opposite side on account of the presence of a fair degree of obstructive deafness. It must be remembered that when taking the Schwabach test we are taking not alone the bone conduction of the side to which the fork is applied but of the two sides together, to be sure the major portion is contributed by the side to which the fork is applied. This borrowing and contributing of bone conduction from one side to the other must be constantly borne in mind and reckoned with when making the Schwabach and Rinne tests.

The Rinne Test: The Rinne test is designated positive when the air conduction is better (that is longer) than bone conduction, negative when air conduction is less (that is shorter) than bone conduction, and neutral when bone conduction and air conduction are equally well perceived, that is one is no longer than the other.

The same fork is selected in making this test as is used in making the Weber and Schwabach tests and the same precautions are necessary as when making the Schwabach test, as to the position on the mastoid to which the fork should be applied. Besides the same attention should be paid to the amount of pressure with which the fork is applied and also the same care should be exercised in avoiding fatigue of the hearing organ. In addition to these precautions one must be careful to hold the fork in a definite manner and at a definite distance from the meatus while taking the air conduction, at the same time avoiding fatigue after a similar manner as when taking the bone conduction. Concerning the position of holding the fork. A fork held horizontally will not vibrate as long as one held vertically, because when held horizontally gravitational force plays a bigger role than when held vertically. Again it will vibrate slightly longer when suspended

than when held upright. When we consider that there are six directions at right angles to one another along which the sound waves travel, and eight lines at 45° to the first mentioned six lines, which are nodal lines (dead lines) where the sound waves from the primary, secondary and tertiary axes meet, one can appreciate the importance of holding the fork before the patient's ear being tested and the control in that position which permits of the most direct transmission of the sound waves to the tympanic membrane. For instance it is not well to hold the fork before the patient's ear best adapted to permit of direct transmission and before the ear of the control in a position approaching a nodal line.

While the purpose of the Rinne test is to ascertain whether the air conduction is longer than bone conduction (positive) or shorter than bone conduction (negative)) for the sake of increased accuracy, it is well to time the difference, so that when we look at the records of a case and find the Rinne positive 40 seconds we can appreciate the fact that it approaches nearer the normal than another case where the Rinne is but 10 seconds positive. If one is thoroughly acquainted with his fork and with the technique in making the tests it is but one step farther to interpret the findings.

If a case under examination shows a positive Rinne of 40 seconds with a fork that shows the average normal to be 40 seconds, the case may be one that is normal or one in which a neurolabyrinthitis is present. If in addition to the positive Rinne of 40 seconds the air conduction is found to be normal we may feel sure that the hearing is normal. On the other hand if the air conduction is found to be shorter than normal then the positive Rinne of 40 seconds speaks for a disease of the perceptive apparatus. In order to ascertain whether there is a disease of the perceptive apparatus or freedom from disease it becomes essential to determine whether the air conduction is normal, and this is done by comparing the patient's air conduction with the control known to be normal, observing the same precautions noted elsewhere.

As to the variety of Rinne findings, to say the least, they are many and each has a different meaning. To take up a consideration of them all would carry one far beyond the limits of a single paper.

In presenting the paper there was no idea that justice could be done the subject in the brief span of fifteen minutes. The object has been to draw attention to what the writer considers a most important phase of the subject of otology, the value of which is in direct proportion to the technique used, hence the title of the paper "Some Remarks on the Weber, Schwabach and Rinne tests."

1831 Chestnut Street.

ACOUSTIC NEUROMAS.

Dr. John J. Shea, Memphis, Tenn.

The question of acoustic neuroma is so vast that Dr. Harry Cushing has written a book upon the subject. The case which I am reporting is of a bilateral acoustic neuroma, of which there have been only twenty-four reported in the literature.

Acoustic neuromas constitute 7.3 per cent of all verified tumors of the brain in Cushing's series, and out of sixty cerebello-pontile-angle tumors, he found forty-seven of them to be tumors of the eighth cranial nerve. The otologist is the first to be consulted because progressive unilateral deafness of a definite entity is the chief complaint of these patients, and upon us depends the early diagnosis. If they are to wait until general intra-cranial symptoms have developed before a diagnosis is made, valuable time will be lost. The neurological surgeon is prepared to cope with an acoustic neuroma, and when given a case early, can remove the tumor with a restoration of the patients usefulness.

In as much as loss of vision follows the loss of hearing and is an indicator of increasing intra-cranial pressure, a tentative diagnosis of an acoustic neuroma may be made on the following facts: First, progressive uni-lateral nerve deafness until no response may be obtained by the forks either through air or bone conduction; second, progressive recession of vision with choked disc; third, failure to obtain a reaction of the vestibular branch with the Barany tests. Vomiting, staggering, positive Romberg sign and headache constitute late symptoms, and if waited upon before reaching a diagnosis, little can be expected by surgical procedures. No one has ventured to operate before obtaining the first two of the above and with the assurance of the third, localization is definite enough to justify an exploration of the cerebello-pontile-angle.

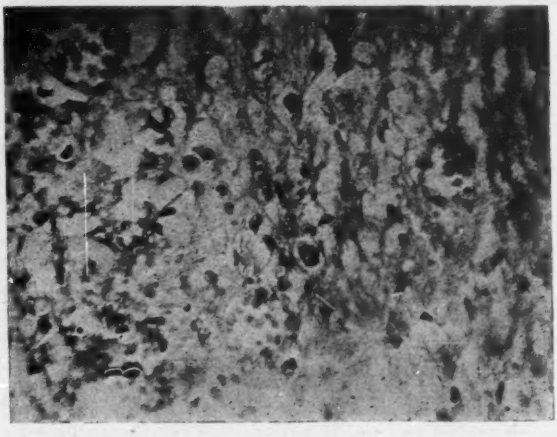
The following case is unusual in that there was a bi-lateral deafness, though it started uni-laterally on the left. After the Barany tests failed to get through any stimulation and all reaction failed, a diagnosis was made of a tumor in the mid brain, which was involving the eight nerves at their decussation, either directly or by pressure. At autopsy we found a tumor springing from each auditory nerve and eight smaller tumors scattered over the brain. Prior to death, the patient was totally deaf and could only distinguish moving objects, but had developed a system of palmargraph, where-

by her mother wrote upon her palm the necessary information, and she would answer by spoken voice.

History: Miss O. M., age 20; referred by Dr. B. F. Turner for a neuro-otological examination.

Family History: Father, mother, one brother, and one sister alive and well. No history of tuberculosis, epilepsy, tumor or insanity.

Past History: Normal birth and progressed naturally until 1915, when she had an acute illness complicated by a right wrist and ankle drop, which was probably an anterior polymyelitis. During the summer of 1918 she complained of drowsiness, and spent most of her time in bed, but recovered sufficiently to attend school the following term. An acute infection of influenza interrupted

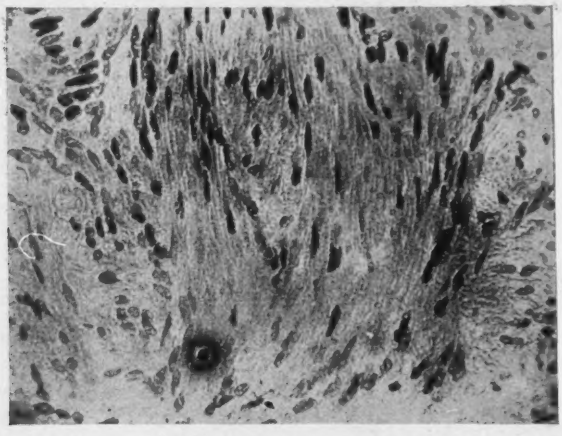


school during January, 1919, for one week. The spells of drowsiness returned during the summer of 1919, but again cleared up before school opened.

Present History: Patient first noticed that the hearing was less acute in the left ear prior to entering college September, 1919. This progressed rapidly and in a month's time, the right ear began to grow deaf. The progression was so rapid that she was required to stop attending the lectures before the end of the first semester. At this time her vision was good and there was no other symptoms. But the vision became blurred in February 1920, and with the blurring she developed an occipital headache.

Chief Complaints: Absolute deafness in both ears; reduced vision; right ankle and wrist drop.

Physical Examination: General appearance is that of a well-developed and nourished young lady. Mentally alert and answers questions with precision. Head is of normal contour and no scars are present. Eyes (Dr. E. C. Ellett): Pupils are dilated, but equal and react to light and accommodation. Vision, O. D. 15/30 O. S. 15/25. There is a choked disc of 2 mm. with normal fields. Nares: post-nasal discharge and congested nasal membrane. Tonsils: were buried but clean. Ears: The membrani tympani were normal. The functional examination showed complete nerve deafness as she could not hear any of the forks by either air or bone conduction. The Galton whistle could not be heard and the Barany Noise apparatus failed to register. Heart, lungs and abdominal organs were normal. Extremities: There was a right ankle and wrist drop.



Neurological (Dr. B. F. Turner): Flaccid paralysis with atrophy of the extensors and external rotators of the right forearm. Weakness with atrophy of the peroneal muscles of the right leg. Sensory: There are no areas of anesthesia or paresthesia. Reflexes are normal save for right wrist and ankle drop.

Barany Tests: Summary: spontaneous was negative. Rotary: appreciated that she was turning, but had no after nystagmus or vertigo. Caloric failed to produce any reaction. Laboratorv Test: Urine and blood were normal—Wasserman was negative on blood and spinal fluid.

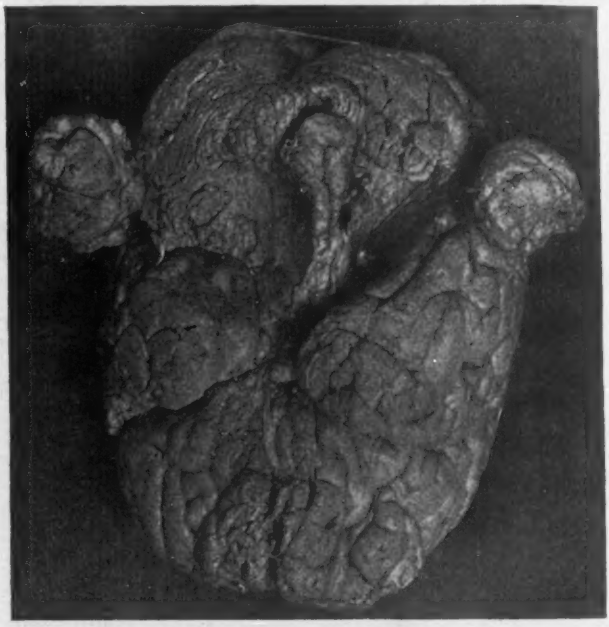
PROGRESS.

March 3: Working Diagnosis: Intra-cranial tumor in the mid-brain pressing on the pathways of both nervi acustica. Advised relief of intra-cranial pressure to save vision.

March 27: Dr. R. E. Semmes did a left subtemporal decompression. The dura was tense and the brain bulged. The descending horn of the left lateral ventricle was tapped and fluid escaped under great pressure. A lumbar puncture was necessary to reduce the pressure sufficiently to close the dura.

April 8: Discharged from the hospital after an uneventful operative recovery with slight improvement of vision, but no improvement of hearing.

October 8: A second Barany examination showed no change in the reactions. October 18 readmitted to the hospital with vision reduced to light perception and moving objects. Patient has had an intensive anti-syphilitic treatment. Dr. Semmes injected the ventri-



cles and the radiogram showed the skull to be very thick and that there was an internal hydrocephalus. The internal auditory canal was dilated. This decided the question that the tumor was sub-tentorium and so on the 20, Dr. Semmes did a bilateral sub-occipital exploration and uncovered a tumor in the left cerebello-pontile-angle. The operation had to be abandoned because the patient went bad and it was hoped to remove the tumor at a second operation.

October 22: Vision improved to recognition of individuals.

October 25: Patient spent the morning on a backrest and was thought to be progressing favorably, but during the afternoon developed a pulmonary oedema and died.

AUTOPSY FINDINGS.

Miss O. M. Case Number.

There was a healed scar in the left temporal region and a healed "Cross Bow" incision in the sub-occipital region.

The skull was very thick and dense; and the porus enternus enlarged on the left, the dura was studded on its inner surface with small, soft apaque white tumors, varying in size from one-fourth to one c.m. in diameter. Eight of these caused marked indentation of the cerebral cortex.

There was a hard encapsulated tumor in each cerebello-pontile-angle, measuring two and one-half c.m. in diameter on the right and three and one half c.m. in diameter on the left. The surface was smooth and the tumor on the left side presented a bossy contour. On the left side there was an elongated bulbous enlargement of the eighth nerve, measuring about one-half by one-fourth c.m.

There was marked compression of the pons and medulla greater on the left, distortion of the lobes of the cerebrum and cerebellum, and marked herniation of the cerebellum into the foramen magnum.

The third and lateral ventricles were enlarged and the lumen of the infundibulum dilated.

Microscopic examination showed the typical picture characteristic of dural fibromata (neuro-fibroma). Many of the smaller growths near the superior longitudinal sinus showed calcarenous deposits.

Permission for general autopsy not obtained.

FINDINGS.

Skull very thick, multiple dura-fibromata indenting cerebral cortex, bi-lateral cerebello-pontile-angle tumor and bulbous enlargement of the left eighth cranial nerve. Internal hydrocephalus (secondary). Compression of pons and medulla and distortion of the lobes of the cerebrum and cerebellum, with herniation of cerebellum into foramen magnum.

A DICUSSION OF MENINGITIS.*

DR. S. MACCUEN SMITH, Philadelphia.

It gives me pleasure to accept your courteous invitation to present, this evening, the ever-interesting subject of otitic meningitis for a round-table discussion. In so doing I am conscious of the fact that little I have to say is new, and yet I hope that some of my statements are sufficiently radical to elicit an animated discussion, which, after all, will be the really illuminating feature of the evening.

The simple, serous form of otitic meningitis, and circumscribed, or extra-dural abscess, will not be dwelt upon, except to emphasize the important point that the average case of meningitis is primarily one of these types; hence the vital necessity of its early recognition with the view of arresting its progress and preventing the establishment of the later stage—diffuse suppurative meningitis—which is almost always fatal. This, to my mind, is of the utmost importance and offers our only hope in most instances for satisfactory results.

Three cardinal points are of fundamental importance if we are to be successful in our treatment of even a small percentage of these cases. The first of these is early diagnosis. This can often be accomplished by examination of the spinal fluid, which will show an increase in the globulins and cell count; anything above ten of the latter should be looked upon as suspicious and, in the presence of other symptoms, justifies immediate mastoid operation. The second is arrest of the circumscribed variety, thereby preventing its becoming diffuse. Herein lies our great hope, and its actual realization happens so often that we can accept it as an accomplished fact. The third point is the employment of surgery to any extent indicated in individual cases, for, after all, otitic meningitis is strictly a surgical disease, and non-recognition of this fact on the part of the general profession has been most unfortunate and at times disastrous. The only safe course to pursue in otitic meningeal disturbances is to regard every such case with the amount of gravity it rightfully deserves. To feel secure in the thought that we are deal-

*Read before the Section on Otology, New York Academy of Medicine, May 13, 1921.

ing with a serous meningitis, and therefore a condition of little consequence, is a mistake frequently made.

Quite recently a patient suffering from an acute otitis media was said to have also a meningismus or meningeal irritation; in another case the otitic complication was pronounced serous meningitis. Complete assurance prevailed as to the progress and condition of each patient for about six days, when they rapidly grew worse, the first alarming symptoms being rigidity of the neck and diplopia. At this stage I saw the cases, each one quickly lapsing into coma and death.

The point I wish to emphasize is that serous meningitis, meningismus, meningeal irritation, or by whatever name one wishes to designate an otitic intracranial inflamatory process involving the meninges, any and all must be accepted as the primary or early stage of a diffuse suppurative meningitis and treated as such if we hope to reduce our mortality. There is every reason to believe that if the two cases cited above had been so accepted, an early diagnosis and prompt operation might have been successful.

For early as well as later diagnostic purposes I place the greatest confidence in an examination of the cerebro-spinal fluid taking into consideration any abnormality of pressure when withdrawn. If we keep in mind that from three to five small lymphocytes, as well as a trace of albumin and sugar and some inorganic salts, together with a slight alkalinity, are characteristic of normal spinal fluid, we can readily note any pathologic deviation. The spinal fluid may vary from simple turbidity to a dense cloudiness, with actual pus. The cell count may be from thirty to fifty only in the early stages of the disease, but thousands may be found as the condition advances. It is important to remember that the cells are mostly polynuclear leucocytes and that the spinal fluid has an acid reaction in meningitis, in contradistinction to its slight alkalinity in health.

Presence of bacteria in the spinal fluid definitely establishes the diagnosis of meningitis. An initial negative result, however, is unimportant in the presence of other characteristic findings and positive results will be found in subsequent examinations. An increase of more than ten leucocytes of the mononuclear variety, together with a substantial increase in the polymorphonuclear percentage, should make one suspicious of a developing meningitis complicating existant aural disease.

The prognosis of otitic meningitis is not governed so much by the clearness or turbidity of the spinal fluid as by the presence or absence of bacteria, together with the intensity of their virulence.

The clinical symptoms in a meningitis due to the streptococcus mucosus capsulatus are frequently absent or appear just at the terminal stage, hence the necessity of an early spinal fluid examination in suspected cases, more particularly in the presence of continuous headache.

Except in children, convulsions are rare in any stage of diffuse meningitis. Their development, therefore, would lead one to suspect the presence of a brain abscess.

Although diplopia is found in other conditions, it has been such a constant and marked symptom in some of my cases that I am constrained to look upon this eye symptom as being of considerable importance. In the only case of what seemed to be a diffuse meningitis that I have ever seen recover, this symptom was well marked before the onset of unconsciousness and continued for several weeks after operation and during convalescence. The records do not show whether the third, fourth or sixth nerve was involved. I would be always on the lookout for a developing meningitis if diplopia were present, and I am also suspicious of a pending meningitis where a patient suffering from aural disease shows a progressive intolerance to light.

Inequality of the pupils, with paralysis of one or more of the ocular muscles, more particularly of the external rectus, are the more common eye symptoms. When the cerebellum is involved, ocular paralysis occurs. Widely dilated pupils are common, of course, in the terminal stage.

In the latent type the mentality is usually normal, whereas irritability and restlessness are common in the more active form. In both, drowsiness and delirium precede the terminal stage of coma.

The temperature in the acute fulminating type is generally high, but the absence of this symptom is of no significance in the early stages of the latent form.

Ordinarily the pulse is rapid, with good volume, until the terminal stage, when it becomes weak and thready, and respiration is slow when fluid fills the ventricles, simulating brain abscess. Respiration is also governed by the stage of the dis-

sease, at first being only slightly above normal, while in the terminal stage it may be the Cheyne-Stokes variety.

The Kernig sign is present in almost every case and therefore very valuable. A positive Babinski is also of value, but not so constant as the Kernig sign.

Broadly speaking there are two distinct types of otitic meningitis. The one develops by silent, slow and treacherous infiltration, frequently without manifesting symptoms until the disease is well advanced. This form of meningitis, on account of its obscurity, oftentimes is not suspected until rigidity of the neck and mental cloudiness appear. The second, or fulminating, type, may force its way rapidly by brutal mass attack, is easily recognized, and terminates in death in an incredibly short time.

Personally I have seen few cases of what might be termed latent meningitis that developed subsequent to surgical intervention. The following case will serve to illustrate what, in my experience, constitutes the so-called latent form of this disease:

E. C. B., a banker, 52 years of age, a giant in stature and strength, suffered from an acute suppurative otitis media, with spontaneous rupture of the membrana tympani, for six weeks before I saw him, during which time slight headache was present. Pain during the first tympanic abscess formation was quite severe, but under treatment by his attending physician the discharge either ceased or materially lessened, some discomfort remaining. Three weeks later this experience was repeated, lasting about a week. In two weeks he again consulted his physician, complaining of discomfort about the ear. This was on a Saturday morning; that evening the physician was called again and found the patient unconscious.

I was asked to see him Sunday morning, he being still unconscious and presenting the various symptoms of meningitis, including marked retraction of the neck and positive Babinski and positive Kernig. He was removed to the hospital and immediate lumbar puncture showed a very high cell count, the absence of sugar and some free pus. An immediate operation was performed, the patient dying in about forty-eight hours.

In a second case meningitis developed in the absence of all symptoms with the exception of a temperature that reached 99° on a few occasions and a more or less constant headache. A diagnosis was made possible later only by an examination of the blood and the cerebro-spinal fluid. In this instance the patient's

friends were naturally amazed when the gravity of his illness was plainly stated, and this not without reason, as the man was, to all appearances, enjoying reasonably good health and was attending to his business even, in a restricted way. The first important symptom was the development of a scarcely noticeable convergent strabismus, together with an occasional diplopia. He had suffered from an acute otitis media eight weeks previously, which entirely cleared up in less than two weeks. In the meantime he spent about four weeks in the mountains, which improved his general health, until the beginning of the last week, when the above mentioned symptoms developed, together with some increase of head pain. During the last week a distinct drooping of the superior-posterior wall developed, and it was almost wholly due to this indication that I was able to secure an immediate operation on the day that he returned from his vacation. It was imposible to elicit the slightest tenderness on deep pressure or percussion over the mastoid process, owing to a thick cortex, but at operation the mastoiditis was found to be of the hemorrhagic type, with some free pus at the tip. The osseous structures separating the sinus and dura were intact and apparently healthy. For two days following the operation all symptoms disappeared except the diplopia. At the end of this time the temperature began to rise and the strabismus reappeared in a very marked degree, while an examination of the blood and spinal fluid and the appearance of convulsive movements all demonstrated the progress of the disease, the patient succumbing four days later. The fact that there was no carious erosion exposing the dura would indicate that the extension to the interior of the skull was through the small vessels connecting these two cavities. An ophthalmological examination did not show any changes in the fundus until one day before the patient's death.

Illustrating the fulminating type is the case of H. B., aged 45 years. I found him unconscious, with retracted head, eyes partially open, and markedly stertorous breathing, offering the following history: In the evening of the second day previous he had had a slight left-sided earache, which he thought due to scratching the external auditory canal. This pain was relieved by the installation of warm boric solution. Without symptoms a slight serosanguineous discharge made its appearance on the afternoon of the following day. Later, at midnight, he devel-

oped suddenly an intense general headache, with great restlessness and convergent strabismus. The attending physician administered one of the coal-tar products and the patient fell asleep. His stertorous breathing the following morning awakened his wife, but the patient could not be aroused. It was then that I was asked to see him, had him removed to a hospital and performed a mastoid operation. The cortex was exceptionally hard and thick. The underlying cells were broken down and bled freely. Free pus was evacuated from the mastoid antrum. There was a carious exposure of the dura through the tegmen antri and tegmen tympani. A decompression operation with drainage, after Crockett's method, was done. The patient died in thirty-six hours.

An examination showed the cerebro-spinal fluid to be under pressure, with high cell count, absence of sugar, and the presence of serum-globulins. A blood examination showed a leucocytosis of 19,400 and a polymorphonuclear percentage of 89. No ocular changes were observable in an ophthalmologic examination. Cultures showed a pure pneumococcic infection of the spinal fluid and mastoid process, and pneumococcic and streptococcic infection was shown in secretion taken from the external auditory canal. Although it would be unusual to subject the patient to an earlier lumbar puncture than was done in this case, I feel confident that the findings here would have demontrated the presence of meningitis two or three days earlier.

After years of more or less discord growing out of diligent efforts and research to master this disease, we settled down to a harmonizing consideration of the subject and concluded, among other findings, that diffuse, suppurative otitic meningitis is, for the present, at least, incurable. Furthermore, we now believe that all the meningeal disturbances, whatever their name or however mild in form, constitute the primary or early symptoms of actual meningitis, and if not arrested by timely measures or by nature limiting the process, will eventually become diffuse and suppurative, with the usual fatal result.

We know that the milder forms of meningitis, and the circumscribed variety, are curable and are being successfully treated in greater numbers every year, simply because we are better able to conclude an early diagnosis and employ prompt measures for relief. I believe frequent examination of the spinal fluid will determine not only the presence of meningitis, but indicate

its progress, just as surely as the repeated caloric test will indicate the progressive death of the labyrinth, when invasion of the meninges occurs through the internal ear.

We are justified in promptly opening the mastoid process in all cases of otitic meningitis, as well as in those cases where a definite diagnosis has not been made, but the disease is suspected. An immediate operation is especially servicable in children. It is not uncommon to relieve a child presenting symptoms of advanced meningitis, with ocular involvement even to the extent of blindness, as reported by Dench, by prompt surgical intervention,—lumbar puncture, mastoid operation and perhaps subtemporal decompression and subdural drainage when indicated.

Our first step in the surgical treatment of meningitis is to eradicate the primary focus of infection, and this requires a complete mastoid operation, simple or radical. The next step is the relief of intracranial pressure, as yet quite incompletely attained by decompression and opening the dura. With this accomplished, we would be able to cure numerous otherwise hopeless cases, but the anatomic and physical difficulties are insurmountable except for the more or less ineffectual spinal route.

The Jansen-Neuman operation is indicated when the infection occurs through the labyrinth. This procedure provides the best location for drainage of the subdural and subarachnoid spaces on account of the deep recess between the pons, medulla and cerebellum. My experience in this operation is practically nil, but, as above stated, anatomically it offers a location in which the brain is not immediately pushed into the dural wound, which in turn actually prevents drainage.

Although early muscular stiffness is frequently observed, I am convinced that pronounced retraction of the neck is a late sign, indicating an advanced stage of meningitis, analogous to a perforated cortex in mastoid disease, or an organized thrombus of the jugular in sinus thrombosis. We have long since abandoned the practice of awaiting the development of the latter two symptoms before instituting surgical intervention. Our diagnostic ability does not have to assume the authority of a prophet to pronounce a condition meningitis where the patient presents even a moderate degree of opisthotonos. When possible, a diagnosis should be made before pronounced musclar rigidity appears and I believe that this generally can be accomplished.

The serum treatment of otitic meningitis has been attended

with little success. Some cures have been reported from intraspinal injections following withdrawal of spinal fluid, as well as the adoption of other surgical measures, so that reasonable doubt as to the efficacy of the serum, per se, must exist. The following case illustrating this point occured in my service at the Germantown Hospital only three months ago: G. L. D., male, 49 years of age, recovered from a double pneumonia, and three days later re-entered the hospital suffering from an acute suppurative otitis media of the right side, with temperature of 104° and some tenderness over the mastoid. A myringotomy was immediately performed, which relieved the severe pain. A lumbar puncture showed the cerebro-spinal fluid to be cloudy and under pressure. Extending over a period of three days, 175 cc. of polyvalent anti-pneumococcic serum was given, 125 cc. intravenously and 50 cc. intraspinously following a lumbar puncture. Unfortunately, the examination of the spinal fluid was very incomplete. On each occasion, however, it was under pressure, cloudy, and contained albumin and sugar. The report further states that no micro-organisms were present. This statement was doubted by the chief bacteriologist, who did not have an opportunity to confirm his opinion, another lumbar puncture being thought unwise in view of the patient's convalescence. When I saw the case Babinski and Kernig signs were negative, the patient was unconscious and the head retracted. The clinical picture was the same that I had seen in many cases which had resulted fatally and before operating I predicted that the patient would not recover. Previous to operation the blood count showed a leucocytosis of 22,300, with a polymorphonuclear percentage of 84. A mastoid operation was performed, the cells being well filled with greenish-yellow pus; no exposure of dura or sinus. Following the operation, the leucocytosis dropped to 14,700 and the polymorphonuclear percentage to 70. Patient was discharged as cured five weeks after the operation. It was impossible to state, under the curcumstances, what therapeutic agent or agents were actually responsible for the recovery. The probabilities are that the man suffered from a limited involvement of the meninges.

We must of course be careful not to confuse the brilliant results obtained by the use of the anti-meningitis serum of Flexner in the treatment of epidemic cerebro-spinal meningitis due to the meningococcus, with that of otitic meningitis. It

would seem that some of the reported cures have been of this type.

In view of the fact that some discussion has arisen as to the possibility of inducing meningitis under certain circumstances, by the withdrawal of spinal fluid, I should like to ask if any of those present have had this experience. Personally I have never seen a case where I had any reason to suspect that lumbar puncture caused a meningitis, and yet we are told that lumbar puncture performed when the pneumococcus has invaded the blood stream does induce a meningitis. It has been further stated by Eagleton, quoting Weed, of Baltimore, that in certain forms of septico-pyemia, notably that from Friedlander's bacillus, a lumbar puncture will unfailingly give a meningitis.

REMARKS ON THE PSYCHOLOGY OF DEAFNESS.*

DR. THOMAS HUBBARD, Toledo, Ohio.

I am speaking for colleagues when I say that we are meeting with the League tonight because, knowing something of the effects of deafness, we duly appreciate the admirable service of this group of men and women organized to aid each other in overcoming the obstacles which the deaf encounter. You have all developed a philosophy of your own and it may seem rather presuming for anyone to attempt to generalize along the line of physchology before those who do understand through actual experiences. A few ideas are ventured with the expectation that they may serve as suggestions for general discussion.

The special senses enable man to properly correlate his life to his environment. Life itself is a continuous adaptation and all of the sense organs play the important role in his relations to things external. Sight, hearing, sense of smell and taste, tacile and muscle sense including pressure, heat and cold and the complex refined sense of equilibrium foster, protect and contribute to make the life complete.

And then we should add those less familiar internal functions such as the appetites, not confined to digestive organs but including the physiologic desires, the pyschic cravings, the gratifications of

*Read at the meeting of the section of Oto-Laryngology of the Academy of Medicine at the Club Rooms of the League of the Hard of Hearing, Toledo, Ohio, Oct. 28, 1921.

expending stored up muscle and nerve forces in wholesome physical exercise, play and work—all included become the expression of adaptation of the animal in response to the stimulus of the environment.

Contentment is simply evidence of harmonious response to the exigencies encountered in life and it implies wholesome realization of conscious and subconscious desires.

The eye and ear are the most easily studied—the most refined and apparently the most essential. It is natural to exalt these special senses unduly and assume that they are indispensable but more careful analysis convinces that above all of these is that supreme function which they all serve—the psychic entity which dominates the whole being, receives and perceives the contributions of the special organs. From this central point we may observe the special senses by a sort of retrograde analysis. That is to say, we determine the true function of a special sense by studying the effects of permanent impairment of the same—the effect on the other special functions, on mentality and disposition.

Deprivation of a special sense is a severe test of adaptability. The philosopher meets it by taking stock, as it were, of what is left to him to aid in successfully striving toward the goal, contented well-being. Strength of character is tested and the real compensation is that the same is fortified by the trials which this handicap entails.

I mentioned the various special functions which, working in harmony, develop a more or less perfect adaptation to environment. When one of the more important fails, sight or hearing for example, there must be a readjustment of all. The psychic function is of course the most affected and reorganization is in order. Let us make a comparison which is in a certain way rather logical. A well ordered household—a family—suffers bereavement in the loss of the father, the provider and protector. Reassignment of the duties of the various members is necessary. Service is exacted from all and accomplishments are forced, the younger members responding and rapidly developing latent talent. That is what happens when the sense of vision is lost. The tactile, the muscle sense, the equilibrium function, orientation by which a man feels his relation to objects and direction—all are toned up and contribute to make up for the loss. We find a very appropriate comparison in the part that hearing plays in the body bereaved of vision. Hearing carries the burden, comparable to the mother taking the place of the father. The ear develops refined faculties and becomes the protector as the dominant special sense.

Let us carry this analogy further in a reciprocal comparison and then into the realm of psychology. A family loses the maternal caretaker. There is a reassignment of duties. The father carries the burden and very successfully perhaps in so far as material welfare is concerned. He is Vision and provides well and protects. The minor special senses do their part, but *there is something lacking in the moral status of that* family—indeed the morale has suffered an enduring shock. Now we are deep in the psychology of deafness. Hearing has a very intimate relation to emotional centers. There seems to be a very logical analogy in this personification of Vision and Hearing and we will develop it later as we work up to the perfection leads to harmony. There is lost energy in cacophony the loss of one was the occasion for this little domestic digression.

We will now consider more in detail the effects of loss of hearing. The spoken words, carried to the ear in modulations, which are often more expressive than the meaning of the sentence, incited and stimulated the evolution of the human brain more than any other one factor. Language itself develops euphoniously because perfection leads up to harmony. There is lost energy in cacophony —waves annul each other—and in a refined language there is a tendency to eliminate harsh combinations of consonants. In tests physical grace of movement is strength conservation. The human voice carries force and conviction when it conveys the proper words in tone and inflection adapted to the meaning.

Deafness deprives one of that fine sentiment as expressed in the well spoken sentence. The music of language is lost. Forced speaking destroys this equality. Consonants are unduly emphasized and modulation is lacking. The deaf must find new avenues of reception of the sentiment conveyed by accent and inflection. The normal mind craves sentiment in varying degree. This is probably one of the greatest deprivations of deafness—they miss the vitamines of daily mental pabulum.

The evolution of music, in so far as the human voice is concerned, is but an elaboration of modulation and here we lead up to the conception of what it really means to be deaf. Music is a closed book. Fortunately it does not depress all in the same degree for about half of us are tone deaf naturally (meaning that half are not susceptible to the thrills of refined music.) But the fact stands that they have lost one channel of reception of sentiment. Emotions are a natural outlet for their suppressed feelings and deafness seals this flood gate.

The intense study of facial expression is an attempt to find a suggestion of the sentiment conveyed by modulation as well as the meaning of the sentence. They study not the lips alone but the whole face, mouth, eyes, eyebrows, even wrinkles and dimples, head poise and gestures, keenly searching for sentiment, and they do succeed in a marvelous way. They become expert judges of character from that kind of practice.

Do not confuse sentiment and sympathy. The deaf person who hunts for sympathy is hopeless or to put it another way, they become very selfish.

The philosopher falls back on other resources. He can read into the printed or written word a deeper meaning. A keen intellectuality comes from this practice. Solitude is not without compensation. It develops character and a penetrating mind. He can cultivate a purely intellectual substitute for ordinary sentiment.

Now all this explains some of the characteristics of the psychology of deafness. The cause of the tendency toward the depression and morbid reflection is apparent. But we are encroaching on private ground. They understand this whole subject better than any outsider but I will only venture the hope that something has been said that will fit in with and help the individual philosophy and if so we have not trespassed unfeelingly.

As in every subjective contest, and it applies as well to the physical, the evidence of and a dominant factor in success is mental balance and good nature. Cheerfulness is essential to bodily health and is fundamentally an evidence of a sound philosophy. It is likewise the visible evidence of winning the game in spite of handicap.

Harriet Martineaux gives it another appealing attribute. The cardinal rule of conduct, expressed in that beautiful letter which has comforted and strengthened those afflicted with deafness for now nearly a century, makes cheerfulness synomymous with unselfishness. Basking in sympathy engenders an egotism that becomes supreme selfishness. Such precepts are intended for the consideration of not only the deaf but for all. To the associates it says—give due expression of sentiment but never excess of sympathy. To the deaf it says—do not court sympathy for fear of the reaction causing selfishness, and maintain that attitude of cheerfulness which makes the life of associates the better for having had the privilege of contact with the self made philosopher.

Just a few words about worry. A proper conception of this antagonist of contentment aids in the banishment of this exhausting

habit. Worry is a cousin or rather is born of fear. The habit of fearing is cowardly. There you have the proposition in two sentences and perhaps it would have been more effective if I had made this the text.

DEAFNESS.

WALT MASON.

My ears don't work the way they should;
My hearing isn't extra good;
And agents come most every morn
To sell some patent audihorn,
Some strange contraption painted blue,
To make me hear as well as you.

I shoo said agents from my door
And tell them to come back no more;
To buy such traps I'd be a loon;
My deafness is my greatest boon.

The fellow with a weary tale
With fungus on it, it's so stale,
Will pass me by before he'll tell
His story when he has to yell.

I miss so many tales of woe,
So many chestnuts all men know,
So much of gossip, mean and punk,
So much of scandalmongers' junk,
That I'd despise the meddling man
Who brought my hearing back again.

And when I seek my couch at night
I'm like a child, I sleep so tight.
The noise that keeps you all awake
My gentle slumbers cannot break.

I do not hear the rounder yell,
I do not hear the milkman's bell;
The chugging motors scorching by
Can't make your uncle bat an eye.

I'm satisfied the way I am;
You see me merry as a clam,
And if I heard as well as you,
No doubt you'd find me grim and blue.

—Read by Mrs. Dewey.

REPORT OF A CASE OF TEMPORO-SPHENOIDAL BRAIN ABSCESS FOLLOWING ACUTE MASTOIDITIS.*

DR. RALPH ALMOUR, New York.

S. O., age 53, male, married, came under observation June 14, 1921, complaining of excruciating pain in the fronto-parietal region. The pain was constant in character, and most intense at night. He suffered from nausea, also, and the night before coming under observation he had vomited.

Patient had a subnormal pulse and had vertigo, particularly when the pain was most intense. He was admitted to the hospital for observation and treatment; and the following history was elicited:

He had had a mastoidectomy performed on his right ear five months before his admission at the Post-Graduate Hospital. For two months he was well; and then he developed an abscess on the right side of the neck which discharged pus until two weeks before admission, when the pains of which he now complained began, radiating from the right temporal region. These pains interferred with sleeping. He had an attack of vomiting two days before admission.

Examination shows that his cerebration is disturbed. Patient fails to concentrate and occasionally fails to recognize people. His vision is disturbed. He seems a well nourished man, presenting a scar over the right mastoid region and another behind the right sternomastoid muscle, which has a sinus in the center of it. His lung, heart and abdomen are negative.

On the fifteenth, examination reveals his pupils to be equal, regular and react promptly to light and accommodation. Abdominal reflexes are absent. Knee jerks and ankle jerks very active. Plantars normal. Gait normal. No Romberg. Finger to nose tests normal. Patient has a fine tremor of hands and tongue. The pain in the right frontal area seems neurological in type.

Urine is negative. Blood count shows 5,600,000 red blood cells, with a hemoglobin index of 60 per cent. White blood cells 8,400, with 82 per cent polynuclears and 18 per cent mononuclears. Wassermann negative.

Blood chemistry is as follows: CO_2 46 per cent. Gl. 0.185 per cent. Urea 12.38 mgs. NPN 23.5 mgs. Creat. 1.8 mgs.

*Read at the Section on Otology, New York Academy of Medicine, Nov. 11, 1921.

Lumbar puncture gave 20 c.c. of clear fluid under increased pressure. Albumen and globulin present. Cu reduced. Four cells per c.cm., all lymphocytes.

Fundi normal. Fields of vision normal.

The opinion of those who saw the patient at this time was that he was suffering from an intracranial condition; but in the absence of localizing signs, no definite diagnosis could be made except the tentative one made upon admission—that of a temporo-sphenoidal abscess.

The headache was complained of all the time and the patient gradually became irrational. On the twenty-third, the right side of his face became swollen and the patient became markedly cyanotis. There were still no signs of localization.

On this day the right pupil showed larger than the left, and was fixed to light. There was loss of sensation on the left side of the face—hemianalgesia—and reduced sensibility of the left side of the body. There was also paralysis of the left side of body. Knee jerks equal. Bilateral clonus present. Babinski on left. Abdominal reflexes were absent.

During the entire time the temperature ranged between 98.8° and 100°. Respiration 22; pulse 62-72.

A diagnosis of temporo-sphenoidal abscess was now made with some degree of certainty, and operation was agreed upon. The patient was taken to the operating room, but died of respiratory failure before operation.

Post-mortem examination showed an abscess in the right temporo-sphenoidal lobe extending from an area near the tegmen celluli upward to beyond the motor area. The dura overlying the tegmen celluli was adherent to it. Dehiscence in tegmen celluli back of external root of zygoma due to surgery upon mastoid at previous operation. Lumbar puncture performed post-mortem gave clear fluid under pressure.

Comment: This case is presented, first, to show the development of an abscess to quite a large extent without focalizing symptoms and, in fact, with symptoms more from the frontal lobes than from the temporo-sphenoidal; and second, to show, in contrast to the slowly developing earlier stages, the rapidity with which the terminal stage of the abscess developed.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

February 10, 1922.

An Interesting Case of Otitic Sinus Thrombosis. Dr. J. L. Maybaum, New York.

E. G., male, 28 years, was admitted to the Manhattan Eye, Ear, Nose and Throat Hospital, service of Dr. T. P. Berens, on March 26, 1921, with the following history:

Previous aural history as regards right ear, negative. The left ear has been discharging for ten years. Three weeks before his admission to the hospital he had a submucous resection of the septum performed. Twenty-four hours later the patient began to complain of pain in the right ear. Myringotomy was done by his family physician one week later. Following this the ear discharged moderately. The pain, however, over the right mastoid, temporal region, and in the ear continued. I was informed by the patient that during the week preceding his admission to the hospital he had had three distinct chills followed by high temperature rises. The last chill had occurred the night before. The patient appeared to be extremely ill; he looked thoroughly septic and was in a semi-comatose condition. Temperature on admission, 105; pulse, 96; respirations, 20.

Examination of the left ear: O.M.P.C., large central perforation; no evidence of acute exacerbation.

Right Ear: Moderate muco-purulent discharge from middle ear; the canal wall was not sagging; fullness in the posterior auperior quadrant, small perforation in this quadrant. Hearing moderately impaired; tenderness and edema over the right mastoid especially marked over the emissary and posterior margin of the mastoid and in posterior superior cervical triangle. No spontaneous nystagmus; pupillary reaction normal; no clinical evidences of meningitis. Decided tenderness at the angle of the jaw on the right side on deep pressure.

While the diagnosis of sinus thrombosis was quite evident from the clinical picture, in order to exclude any accompanying conditions or complications, thorough laboratory and physical examinations were made.

Blood culture report twelve hours after being taken showed hemolytic streptococci infrequence; seven colonies in six blood plates showed: W.B.C., 15,4000; Polynuclears, 96%; Small Lympho., 3%; Large Lympho., 1%.

X-ray Report: Right mastoid, large cells filled with granulations and pus with no bone destruction.

Heart and Lungs: Negative.

Urine Examination: Slight trace of albumen; sugar, negative. Few granulation casts, few pus cells.

I did a simple mastoid, ligated the jugular vein and opened the lateral sinus. On removing the cortex, the underlying cells were found to contain granulations, but no free pus. There were areas here and there of softening. The sinus plate, as far as macroscopical changes were concerned, appeared to be normal. I rapidly uncovered the sinus plate with a gouge and found the sigmoid sinus definitely diseased. Exposure was carried back toward the torcular for a distance of two and one-half inches before normal sinus could be seen. The lower end of the sigmoid sinus had a decidedly yellowish, grayish appearance. The jugular vein, apparently normal, was ligated directly above the omo-hyoid. Returning to the mastoid wound, the sinus was incised and an extensive, firm, obturating clot from the bulb to about two inches posterior to the knee was

removed. Sinus wall excised and free bleeding from the torcular end obtained. No bleeding from the bulb end. The patient being in good condition, I rapidly completed the sinus exposure at the lower end by removing the postero-external wall of the sigmoid sinus to the bulb, removing the jugular process of the occiput as a last step. The completion of this procedure was a matter of an additional few minutes. A clot similar in appearance to that removed from the sinus was curetted from the bulb and free bleeding followed. An iodoform drain was placed in the bulb, the wound left widely open and packed with iodoform gauze.

The patient reacted well to the above operative procedures.

Subsequent Course: Two days following the above operation patient developed a peri-articular swelling, redness and fluctuation over the left sterno-clavicular joint. This I opened the following day under local anesthesia and removed about one-half ounce of pus. Peri-articular swelling developed in the region of the left wrist and right ankle, but subsided in a few days without operative interference.

Following the mastoid and sinus operation, the temperature ranged between 100 and 105 for a period of five days. Even with a rise of temperature as high as 105, and at one time 106, the pulse remained proportionately slow; that is, about 100 and less. On the fifth day the temperature suddenly rose from 101.5 to 106.2; twenty-four hours later there was another rise from 101 to 106. Considerable pus could be squeezed from the neck into the mastoid wound through the bulb. The neck wound showed evidence of breaking down. Patient was taken to the operating room April 3d. I resected the jugular vein. There was a broken-down clot in the vein and a small amount of pus in the upper part of the wound. Dakin's solution through the exposed bulb used for ten days; at the end of this period the wound had a healthy granulating appearance. From the 10th of April until the 18th, the temperature did not rise higher than 102 daily. For a period of two weeks following this there occurred an afternoon rise of 100 to 101, this gradually became normal. On the 14th day of May, I did a plastic operation in order to cover the exposed mastoid area. Subsequent healing uneventful. Patient left hospital on the 21st day of May in excellent general condition. Mastoid wound and neck healed, middle ear dry.

Further laboratory reports:

March 26, 1921: Culture from the blood showed positive hemolytic streptocci infrequences. Culture from right mastoid and clot from the lateral sinus showed profuse growth hemolytic streptocci infrequences.

March 31, 1921: Blood culture, one colony of hemolytic streptocci in six plates.

April 9, 1921: Blood culture, no growth after forty-eight hours.

The case offers the following features for consideration:

1. Following a nasal operation last February, there resulted an acute otitis media, mastoiditis and sinus thrombosis with metastases. The inadvisability of performing non-urgent nose and throat operations during an influenza epidemic suggests itself.

2. A moderately involved middle ear and mastoid with sinus plate hard and intact and a diseased sinus containing an extensive obturating clot, peri-articular metastases, are the important pathological findings in this case.

3. Following a jugular ligation, occasionally need arises for reopening the neck wound and performing a resection. Tobey of Boston, reports statistics in the 1921 "Transactions of the American Triological Society" of two hundred jugular operations, of which but two required resection. Such has been the experience of I. Friesner, J. J. Thomson and other otologists. The rapidity with which a ligation can be performed where the question of time is of extreme importance, the comparative infrequency with which a reoperation is necessary, the localized character of the infection if reoperation is indicated and the insignificant resultant scar are some of the advantages which off-hand suggest themselves in favor of a jugular ligation where the vein is patent and normal in appearance.

4. In the absence of bleeding from the lower end of the sinus, it is advisable to complete the exposure of the diseased sigmoid sinus to give access to the bulb, provided, of course, the condition of the patient warrants a procedure requiring only a few additional minutes. Sufficient exposure is obtained without following the radical procedure of Grunert.

DISCUSSION.

DR. DENCH said the patient went through the operative procedure and was cured. Of course, placing the ligature about the vein above rather than below the facial would have saved the patient the infection, for later it had to be resected and the neck wound re-opened. If you are going to cut off the infection from the general systemic circulation, ligation may be sufficient, but it is important that the ligature be placed around the jugular, above the facial vein, rather than below this vessel. In certain cases where he had taken out the jugular, resected it, metastatic abscesses later developed, but it seemed certain that few of these cases would occur if the ligature were placed above, rather than below, the facial vein. He did not see the reason for ligating and then removing the jugular vein later. Very likely if the ligature had been placed about the internal jugular above the facial vein, ligature would have been sufficient.

DR. PHILLIPS said this was the first record he had heard which furnished any argument whatever in favor of resection. Barring one or two cases, he had not done a resection in five years. Last year he operated on a case in which the ligation was well below the facial vein; the patient was in good condition and in five minutes he carried the dissection well above the facial ligation. He had not heard any history that showed any argument, in his opinion, in favor of resection save this one of Dr. Maybaum's, showing that because this was low down, as Dr. Dench said, it should have been ligated higher up or else resected. The temperature was not even unduly high. Last winter Dr. Goodrich Smith had a case in which the temperature reached 107° immediately after a resection, and the patient made a perfectly good recovery.

DR. MAYBAUM said that his feelings in the matter of jugular ligation or resection had always been that theoretically resection was the best procedure, but the experience of many seemed to be convincing that with a patient in as serious condition, as so many of them are, and the question of time being an important element, ligation was the safer procedure, and that if resection is necessary later it would be a localized condition with which one has to deal. The culture taken in this case 48 hours after the operative procedure showed but one colony. From reading the discussions of this matter in the literature of ten years back, he had found that the opinions were just about as diverse then as now. Those who do resections, however, did not seem to prove their contention that it is the ideal procedure, for many of their patients would have recovered with a simple ligation.

Serous Meningitis Complicating Acute Suppurative Otitis Media; Simple Mastoidectomy; Recovery. Dr. James J. King.

C. H. K., age 45, was first seen by me on November 30, 1921. He gave the following history:

About two months ago was seized with a dull, aching, throbbing pain in the left ear which persisted until about six weeks ago. There was a spontaneous rupture of the drum membrane at that time, followed by a bloody discharge for one day only. The pains in the ear immediately disappeared and he thought no more of the ear, although he had been quite sick, running a temperature between 101 and 103. His tongue had been heavily coated and he had been unable to sleep and was very restless.

Upon examination I found slight redness of the left membrane tympani without bulging and no discharge from the canal. His temperature was 103. He was very restless, tossing around in the bed, com-

plaining of violent headache. His tongue was heavily coated and there was stiffness in the neck. He was sent to the French Hospital for further observation and treatment.

Dr. R. H. McConnell examined him with me and found marked rigidity of the neck muscles. Kernig sign was present. The leucocyte count was 28,000. X-ray of the mastoid showed the left to be cloudy. The examination of spinal fluid showed a cell count of 1000 cells, without growth of bacteria upon culture.

Examination of the eyes by Dr. May was as follows: Pupils normal in size, circular, equal, normal in reactions. External ocular muscles, good motion in all directions, eyes parallel, no paresis of external muscles. Fundi, both absolutely normal.

Upon this evidence we made the diagnosis of subacute mastoiditis with serous meningitis, and advised an immediate mastoidectomy.

The usual simple mastoid operation was done on the first day of December. The cortex of the mastoid and cells were found to be sclerotic and there were areas of granulation in the cells. No pus was found in the mastoid. The lateral sinus was placed forward and was uncovered. It appeared to be much darker in color than normal and it was uncovered throughout its course in the mastoid wound. From its appearance I feared a thrombus. It, however, was finally broken and from the free bleeding I was certain that no thrombus was present. The dura was exposed in the middle fossa and was normal in appearance. Post-operative condition was good.

He was a very sick man and we had fears for his recovery. His temperature for the next few days ranged between 100 and 102, pulse about 90. His severe pain in the head persisted for a few days. He was restless, delirious at times, with twitching of arms and legs, and had to be catherized. On December 3rd, he was irrational at times, twitching of arms, pain in back. December 4th, he voided for the first time since the operation. December 5th, more rational, less twitching, some nausea. On December 9th, had chill and temperature 101.4, pulse 100, respirations 32. Very restless. The wound was found to be in a satisfactory condition. On December 15th, his temperature became normal and continued so. On the 18th, he was allowed to get out of bed for 15 minutes. The time of being out of bed was gradually increased day by day as his strength increased. He was discharged from the hospital on December 24th, and came to my office for dressings until the wound was completely healed.

He is perfectly normal now and the mastoid wound has completely healed, and the ear has good function. He hears all the notes, both by bone and air, in this operated ear, but his hearing is somewhat deficient for the voice and the watch.

DISCUSSION.

DR. DENCH said that it was an extremely interesting case, especially in regard to the cell count of the spinal fluid, which was rather unusual. The more one sees of irregular cases of this kind the more he is convinced that we do not yet know as much as we would like. He had seen a number of cases with a similar condition where exposure of the dura had resulted in complete recovery. Such cases teach that a high cell count in the spinal fluid does not necessarily mean that one must necessarily take out the labyrinth or do some very serious operation for a meningitis as many were inclined to think some years ago. Dr. King had not said whether the labyrinthine reactions were normal.

Dr. Dench said that some years ago he had read a paper on the treatment of this condition at the Budapest Congress, in which he recommended exactly the treatment described by Dr. King. No matter what the meningeal symptoms may be, get out the primary focus and then expose the dura freely.

A Study of the Tonal Ranges in Lesions of the Acoustic Nerve and Its End Organ. Dr. L. W. Dean.

The Methods of Measuring the Tonal Range, With Description of the Pitch Range Audiometer and Its Uses. Dr. C. E. Seashore.

DISCUSSION.

DR. ZOPETZKY said that it was very apparent that the study of otology had developed from the study of deafness.

Later on the development of surgery so interested the profession as to overshadow the care of those who are handicapped for lack of hearing. The text-books have preserved the roads on the physics of sound and the function of the ear as originally laid down, but although those who are handicapped for lack of hearing are with us all the time, not much has been done in that line.

It was with the preventive branch of this subject in mind that the opportunity was seized to invite Dr. Dean and Dr. Seashore to present their topic, which is of such great importance in stimulating further study along these lines.

DR. EDMUND PRINCE FOWLER: I appreciate the opportunity here offered of listening to the very complete detailing of the development and uses of the Iowa Audiometer by Dr. Dean and Professor Seashore. To conserve time and to concentrate our attention upon the essentials, I shall read from my notes based upon the study of Dr. Dean's and Mr. Bunce's papers in the 1921 Transactions of the Triological Society and from reading over and listening to the papers read tonight.

The crudeness of our past procedures and the imperative need of accurate means wherewith we may measure the hearing power and aid in the diagnosis, progress and prognosis of ear disease is apparent to anyone who has given a little thought to these matters.

I shall first and mainly discuss the instrument, as the graphs were obtained therewith and depend for their accuracy and significance upon its trustworthiness. The necessity of determining the acuity of hearing at "each and every pitch throughout the range of hearing" is not apparent. How many tones are there within the range of hearing? Even between, say 16 V.D. and 4096 V.D., why take 4000 intermediate tones, rather than 4,000,000 or any other number, unless we interpret a tone interval as one (1) cycle (1 V.D.) per second? The measurement of any great number of tone intervals is out of the question. The extent of the tone intervals necessary to completely determine a curve depends upon: First, the distances between the irregularities or undulations of the curve; and second, the accuracy desired as to the details of these irregularities. For example, take a curve which consists of large undulations, say 1000 cycles long, and of smaller irregularities, say 10 cycles long on the average; then, frequencies 200 or 300 cycles apart would completely determine the curve for the large undulations, but would not detect the small irregularities. In order to measure these latter, it would be necessary to take measurements at frequencies 2 or 3 cycles apart. But if these lesser irregularities are small in amplitude (intensity) compared with the accuracy of observation, then it is superfluous to try to measure them, and readings taken 200 or 300 cycles apart would completely determine the curve.

The frequency intervals test adapted for our measurements can be determined only by experience.

As to the intensity units, whether they are absolute or relative, they *must* be definite, but I am unable to find *what* definite unit, if any, has been used in the pitch range audiometer. The scale does not appear relative to anything in particular. What are the steps used in relative energy?

Purity of tone is of fundamental importance, but as it is impossible to obtain by any means an absolutely pure tone, it is necessary to know the means used to determine the comparative purity of the tones used. If the impurities in a tone are suppressed with sufficient margin of safety so that they do not interfere with the accuracy of our testing, this is all that is required. Again, I am unable to determine how this has been accomplished, if it has been done.

In this connection we note that ordinary telephone receivers are used. Such receivers would at some frequencies (especially below 1000 V.D.) allow impurities to pass with more facility than the fundamental, and this might lead to gross errors.

A continuity of pitch, maintainable at a given known intensity throughout the gamut of normal hearing, is theoretically desirable, but all of the reasons given for its importance do not appeal to me. First, whereas the ear will fatigue rapidly for continuous high tones, it is not necessary to dwell on such but for an instant, if a proper technique is used. Second, to detect true gaps and islands accurately the method is not necessary; in fact, it may be very bothersome because all ears, when measured on a basis of absolute pressure at different frequencies, show such so-called gaps and islands. (I take it that by tone gap and tone island is meant a tone depression and a tone elevation, respectively.) An abnormal ear, then, may differ in this respect from a normal ear *only* in the extent and depth or height of their gaps and islands. Therefore, to have definite significance, the gaps must be abnormally large (say more than an octave and at least one-third deeper than the normal variations). Manifestly, it is not necessary to have a continuous tone series to detect any such.

If the time consumed in examinations can be shortened without sacrificing accuracy, well and good, but with the apparatus described it appears that only at *one* intensity would it be possible to sweep through with accuracy, and even than we are in ignorance as to the exact intensity (force) used. At all other intensities it would be impossible to sweep through with one intensity, owing to the variability of intensity with changes in pitch. This, as I have just stated, may be obviated possibly at one given intensity, but not at all intensities, and in fact, not at any other intensity in the apparatus described. I am convinced that the tonal range can be determined with precision only by measurements made with definite intensities at each and every pitch used. In other words, definite intensities as well as definite tones must be employed.

My criticism of the instrument, technique, and mode of charting naturally implies that I believe Dr. Dean's graphs to be defective. I do so believe, for having neither a uniform frequency or intensity scale, the curves have a very indefinite significance. The frequency scale should follow some law, such as in musical scales, or in a scale of constant frequency intervals. Either of these would mean something definite to the observer. The intensity must be some definite known function of the sound pressure, or sound energy. This also can be done. It appears to me that the form of the curves shown often depends largely upon the characteristics of the instrument. We must guard against such forms leading us into error. Now in spite of the imperfections of construction, and plotting of curves, many interesting suggestions are brought out by the graphs so beautifully demonstrated tonight. These are no doubt apparent to you at once. I use the word suggestions advisedly, because, although our reasoning as to their significance may prove correct, I am of the opinion that after more exhaustive research on the subject it will be necessary to radically change some of our inherited dicta on the acoustics of the ear. We are hardly beginning to fathom many otologic problems, and we do not know what some of our curves mean. The work accomplished by the authors is of the greatest value, and I wish my criticism to be taken, not in any antagonistic or depreciatory way, but as a frank opinion as to how audiometry should and can be made of untold value to us all.

Gentlemen, a new and greater era in otologic practice will ensue from efforts such as those outlined by the authors tonight.

DR. CATTELL said that he assumed that some otologists, as well as most psychologists, do work that is open to criticism; but Dr. Seashore's work was as nearly exempt from any criticism as that of any one in the country. Dr. Fowler had questioned the accuracy of the method of measuring intensity, but it seemed very doubtful if the electric com-

pany to which Dr. Fowler referred had any better method than had Dr. Seashore. Dr. Seashore's method of measuring by actual sight the sounds produced was a great advance. His method of determining the pitch discrimniation, and giving children the chance to avoid defects in the future is most important. In these respects, Dr. Seashore is a leader.

DR. A. MICHAELIS fully endorsed the papers, and said that discussion of a subject of this kind was greatly needed in the section. As the chairman had said, the profession had laid great stress upon and perhaps indulged themselves too much in the dramatic part of the science of otology—the major surgery—and too little has been done for the relief of the deaf person who was the primary consideration of the otologist many years ago. He himself took great interest a number of years ago in this subject, but unfortunately he too had lately let the dramatic part absorb him and had let the other part of the work stand in abeyance; but when he read the program he came to the meeting with great avidity to hear what was to be said Perhaps the treatment of chronic ear cases that come to the attention of the otologists have the worst attention of any, but there is a great difficulty in the way of treating them properly—the enormous amount of time required for the correct diagnosis of their auditory function. It is greatly to be regretted that these cases which would teach so much about the auditory function should be sidetracked in favor of others that come to the clinics

Some emphasis had been laid on the matter of "purity of tone," but purity of tone does not seem to be so very essential in the practical work of testing the hearing, for after all in nature there is hardly any pure tone, if musical instruments are excepted In the human voice there is no absolute purity of tone. It is a composite.

Dr. Michaelis said he was afraid the audiometer was not a very practical instrument in the work of most otologists, and that for a long time to come they would have to continue to rely upon the old Bezold-Edelman series, which is not to be despised.

It was readily seen from the investigations of the readers of the papers how multifarious are the lesions and how frequent are the defects of the auditory nerve which are not ordinarily perceptible.

DR. JOHN GUTTMAN said he had been interested in this line of work for twenty years or more, and that the members were greatly indebted to Dr. Dean and Dr. Seashore for having focussed the attention of the profession on this special line of work, for probably fifty per cent of the patients who consult the otologists are suffering from either defective hearing or tinnitus. The subject is therefore not to be lightly considered.

He agreed fully with Dr. Dean, and could corroborate much of what had been said by the number of cases which he had had occasion to examine with his own accumeter, the description of which had appeared in the last number of THE LARYNGOSCOPE. Affections of the eighth nerve are quite common and are often overlooked by the patient as well as by the doctor. Only by such studies as had been shown here could a correct diagnosis be made.

The statement had been made that some of the cases showed a diminution of bone conduction of three seconds, from which the speaker seemed to draw conclusions. You cannot note well a diminution of as little as three seconds. Before the patient can state he does not hear, before one can strike the stop watch, three seconds have passed; and if the experiment were repeated a few times the results would probably differ materially. Another statement made by Dr. Dean was that he tried to make a differentiation between neuritis and labyrinthitis; he tried to localize the affection in either the nerve or the end organ. Dr. Guttman said he did not know how far such a distinction was possible. Also that when Dr. Dean stated that the removal of tonsils improved or eliminated the affection of the eighth nerve, he could hardly believe such a result was possible.

In quite numerous cases in the charts illustrated this evening tone gaps in the curves were shown. By a tone gap is meant that a patient hears,

e.g., a sound of 1000 vibrations per second, no sound of 1500 vibrations, and hears again a sound of same intensity of 2000 vibrations per second. In his limited experience with his accumeter, Dr. Guttman had not encountered a single case of this type. Might it not be possible that the tone gaps were the results of errors of observation?

DR. SEASHORE said that the hour was too late to attempt to answer all the questions. He appreciated very readily the points made by Dr. Fowler, for they were points on which he had been working for many years. Tonight he had merely stated his conclusions after having worked with both types of instrument; that is, his own and the type that Dr. Fowler has worked with. One of the best forms of the latter is installed in the University of Iowa. For the determining of the upper limit of hearing, the vacuum type principle will undoubtedly be the final instrument and we are using it for that purpose at the present time. But for work within the significant tonal range, the type of instrument described this evening seems to be more serviceable.

DR. DEAN said that he wished to emphasize just two points: first, the examinations upon which the reports were made were carried out by Mr. Bunch. Mr. Bunch devotes his entire time to the testing of the tonal range and he, Dr. Dean, had great confidence in Mr. Bunch's results. He felt certain that Mr. Bunch could measure approximately a decreased bone conduction of three seconds.

Regarding the graph, Dr. Dean said it was determined by Mr. Bunch from the examination of patients who were clinically normal. The curves of all clinically normal cases do not deviate from it in any appreciable way. As mentioned in the paper the telephone receiver used has much to do with the form of the graph. The curve secured from the examination of the pathological case represents approximately the machine. He had confirmed the findings many times with the Bezold series of forks and had every confidence in the findings with the audiometer.

SECTION ON RHINOLOGY AND LARYNGOLOGY.

February 15, 1922.

"Hare Lip and Cleft Palate." Dr. M. N. Federspiel, Milwaukee, Wis.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Case of Congenital Palate. Dr. Henry S. Dunning.

The patient was a young man twenty-one years of age who had never had any operation previous to December. Dr. Dunning made two lateral incisions back of the molars and with a very thin elevator went up as high as he could between the hard palate and the soft tissues into the nose, bringing down the flaps in the opposite way to that of most men. Instead of going down under the flap at the edge of the cleft, he went up and forward and loosened the entire flap without traumatizing the median line edge. The tissue was then brought down and the edge everted and mattressed through, etcetera, and the free edges brought together with interrupted horsehair sutures. A lead ribbon was put in the soft palate after method employed by Dr. MacKenty, paralyzing the palate and preventing the spasm by pushing it up in the center and taking all stress off the suture line. There was no infection and an absolute closure of the entire cleft resulted.

DISCUSSION.

DR. SAMUEL LLOYD said that it has been a great privilege to listen to Dr. Federspiel's paper, and that is it so sound that one hardly knows where to begin to discuss it, but that he would begin at the very beginning. Dr. Lloyd said that a great many years ago when he first began to practice in New York, so many years ago that he did not like to remember, his preceptor and afterwards his partner never operated on a cleft palate. He had tried a great many times, and always with fail-

ure. He was a very successful operator on harelip. A few years before he died a family came to them, and in that family there were seven children—three boys and four girls. The four girls were all normal. The three boys all had double harelip and cleft palate. Dr. Little operated on them, and got very good results. The interesting part about this story is that Dr. Lloyd has since operated on the next generation. The boys in this generation have also harelip and cleft palate, and Dr. Lloyd has heard recently that some of the third generation are on their way to him, and that the males again are showing the same defect. Dr. Lloyd said that perhaps he ought not to have spoken of that, because it is giving away the time he has been in practice, but nevertheless, it is a scientific fact.

A few years ago the Society of Eugenics got interested in the subject and took 250 of the cases Dr. Lloyd had operated on, and investigated their family histories. It is a most interesting collection of cards to see, as we go back over them in how many instances harelip or cleft palate appeared in the ancestors in those cases that it was possible to trace.

The point that Dr. Federspiel made on the experimental work in dogs in which he was able to develop quite a strain of dogs with harelip is another contribution to the fact that these things may be hereditary. There may be a congenital tendency towards defects of this type. The same experiment was carried out by the Society of Eugenics.

Dr. Lloyd was very glad indeed to hear what Dr. Federspiel said of the pre-operative care of these cases. There is no question in his mind that if the proper pre-operative care is carried out the alveolar clefts can be very largely and very rapidly approximated. Dr. Lloyd feels that the great difficulty which most men have in these cases is where the pre-maxillary bone is protuberant. In most of these cases just as soon as you get the premaxillary down you also flatten the nose. Where the premaxillary bone is protuberant the nose is also drawn out of place, and if you pull it down into place you get the flat nose—the African nose. Dr. Lloyd has been in the habit in those cases of taking the skin off the anterior portion of the premaxillary and using it to make the columna nose.

When doing a harelip Dr. Lloyd always brings the nostrils into position in order to make the nose as nearly like the opposite side as possible. There is one other thing to consider. Almost invariably the nose is drawn down and to one side. It does not come down in the median line straight, an it should be brought back in the median line at the time the harelip is done.

The question when to operate is to Dr. Lloyd a very important one, and one which he thinks the individual surgeon must determine for himself. It is impossible to take every case and say it should be operated on in twenty-four hours or in three weeks. There are a number of cases one sees that would not stand any operative procedure at all. The children may be under-nourished; they are not doing well, they have no resistance, and even a minor operation would be a serious affair. The operation must be done only when they reach the proper physical condition. In these cases the surgeon must also take into account the extent of the operation that will have to be done. If a premaxillary has to be brought down then we must have a patient who will stand operative procedures better than the very small operations. As a general rule, Dr. Lloyd wants to get the lips closed before the eruption of the teeth, and consequently he tries in every instance not to let them go beyond eight to ten weeks at the most, but to get them to a point where they will stand operation by that time.

One must take into account whether it is better to operate in one or two stages. Dr. Lloyd believes in many cases that the two-stage operation is much the preferable method.

Dr. Lloyd agreed with the reader of the paper in his idea of closing the lip. He always does that first. He cannot see that it offers any

difficulty whatever in handling the case afterward. When Dr. Lloyd is doing these lip operations he prefers to have the patient held up against the shoulder of the nurse. The nurse sits in front of Dr. Lloyd, and the patient is held up so that the face is directly in front of the operator, and in the normal position when the patient is erect. Dr. Lloyd sees the contour of the face as the patient is going to carry it through life, and exactly as the sculptor works on his block with the block directly in front of him, so Dr. Lloyd works on the lip. He is almost never satisfied with his first suture, but almost invariably readjusts it before he gets through. One other thing is important in the closure of the lip. He wants his suture to go through the skin at right angles and then dip back and catch the muscle and come forward and come out through the mucous membrane just as near the margin as possible, so that in every case he brings the skin and muscle accurately into position so that there is no retraction of the muscle away from the edges. In that way the furrow without any muscle at all is avoided. Dr. Lloyd goes back and catches the muscle on each side and pulls it forward.

Dr. Lloyd was very much interested in Dr. Federspiel's pictures, and in his ideas in regard to palate operations. That he thinks is the most difficult part of the work. Harelips are a comparatively easy proposition; they simply depend upon how much of an artist one is. In that connection Dr. Lloyd wishes to emphasize the fact that he never makes a lateral incision in the lip. Incisions must be made by elliptical incisions lengthening out the short side by the proper elipse on that side, and so getting the two edges exactly alike. One side of the vermilion border is almost always narrower than the other. He brings the lip together where the points of the vermilion border come in contact and then takes the piece he has saved from the cleft, and so gets the same vermilion border on each side. In cleft palate, Dr. Lloyd thinks that is one of the most difficult things we have to do, and it is the most unsatisfactory operation. He thinks he has done all of them, and he has modified all of them. The lateral incision, as Dr. Federspiel says, is unsatisfactory. One gets shortening and pull and then too much tension, and a late separation. These cases give a great deal of difficulty afterward because of the condition of the palate. Dr. Lloyd was glad to hear Dr. Federspiel bring out the idea that he had now reached the point of splitting down through the soft palate, because he reached that point some time ago. He never cuts off the soft palate any more, but splits it. It gives a better approximation, and does not take any tissue away. However, he has always been in the habit of taking the tip of the hamular process off. Dr. Lloyd intends to try the plates demonstrated by Dr. Federspiel, and he thinks it is a great step forward that the orthodontists are taking an interest in this because the loss of tooth buds has been one of the difficulties we have all had to contend with.

Dr. Lloyd does not believe that anyone who tried the Brophy method years ago is satisfied with it. He has seen very few cases where he thought it was well done. If the majority of cases are considered, one will find that if a Brophy operation is done the upper jaw will be narrowed, and an overhanging underjaw will be produced, and teeth destroyed. He does not believe that it can be done without this happening; it may be possible to escape the tooth pulp, but Dr. Lloyd has not been able to do it himself.

One great difficulty Dr. Lloyd had for years was in handling the instruments, and so he had a periosteotome made, and he now uses a right angled needle which answers in every case for the closure of the cleft.

Dr. Robert H. Ivy. I believe the most important thing to be done early in cases of complete harelip and cleft palate is to get the incisive bone back in alignment and closure of the alveolar cleft. This should be done as soon after birth as the patient's health permits. Parents, of course, are very anxious and often insistent about having the lip

closed at once and the visible deformity thereby corrected. If the child is in good condition this may be done at the time the incisive or intermaxillary bone is brought back or the alveolar cleft closed. But in cases of lowered vitality, it is better to put off the lip closure until the child is gaining weight on a suitable formula. In the interval, drawing the edges of the cleft together with adhesive tape, as suggested by Dr. Federspiel, greatly diminishes its width and makes subsequent closure easier. The lip closure should therefore be done as a rule anywhere between the ages of four weeks and four months. The remainder of the palate closure should be made between the ages of six and eighteen months. The importance of preserving the incisive or intermaxillary bone cannot be too strongly emphasized. All of us have seen cases which, operated on as babies, have had the protruding intermaxillary bone entirely cut away, and the lip closed, leaving a horrible deformity with recession of the upper lip due to absence of incisor teeth and alveolar process. In preservation of the intermaxillary bone, I have found difficulty after incising the vomer in holding the protruding mass back in position by a simple wire passed through the vomer. I think it best to pass a silver wire through the anterior part of the maxillary bones on each side and twisting the ends around the front of the intermaxillary bone beneath the median portion of the lip. If the wire is placed high enough it will seldom injure the tooth buds. It is unnecessary to use lead plates in connection with this wire. Various plans have been suggested for bringing out the fullness of the upper lip after loss of the incisive bone or where the latter has been carried too far back. One of these is that of Dr. Brophy, who takes a pedicled wedge from the lower lip and swings it into the upper. Other cases have to wait until complete eruption of permanent teeth and development of the jaw and then have the missing bone and teeth replaced by a vulcanite prosthesis. With recent advances in plastic surgery, there should be some means of replacing this lost tissue surgically. This is merely a suggestion, and the only attempt made by me to accomplish this has failed. The patient was a girl seven years of age, who was born with a complete double harelip and cleft palate, with protrusion of the intermaxillary bone. The surgeon who operated on her shortly after birth cut off the intermaxillary bone and then closed the lip. She later had an attempted repair of the palate, but nothing held except the soft palate. She now has the typical flat upper lip with a large opening into the nose through the incisor region in front and the hard palate behind. There is insufficient palatal tissue to close the opening. I raised a long pedicled flap of skin and subcutaneous tissue from the chest, with its base on the left side of the neck, suturing it back in its original bed for delayed transfer to the mouth. Two weeks later the edges of the palate defect were freshened and the distal end of the skin flap was sutured into it. I hoped thus to close the palatal opening and also build out the contour of the upper lip. Unfortunately, I attempted too much at once and the end of the flap in the mouth sloughed, forcing me to return the remaider of the flap back to the neck. In spite of this failure, I still think the operation of building out the lip by placing a skin flap beneath it to be feasible. The mistake was made in not suturing the end of the skin flap to the freshened tissues under the lip first, and then later, after good circulation from the lip had been established, dividing the pedicle and swinging it back to close the palate. Blair and others have successfully closed palatal defects with pedicled skin flaps from the neck or chest in cases where the palatal tissues were insufficient, but these cases were all in male adults. It would be of great advantage to be able to do this successfully in young children for whom the making and wearing of obturators present difficulties, on account of changes in size of the mouth, eruption of teeth, etc., with growth. I had hoped to report this case tonight as a success, but believe, even as a failure, it is worthy of mention as carrying a suggestion of what may be possible.

For the harelip operation, I prefer the caliper method of Thompson. He measures with calipers the vertical distance from the lower border of the sound nostril to the free margin of the lip, and marks the same distance with the points of the calipers along the borders of the cleft from its upper end to the muco-cutaneous junction on each side. The margin is trimmed away on each side between these two points and then the vermilion borders are incised to make an angle of about 70 degrees with the skin incisions. This gives raw surfaces of equal length on each side to be sutured together.

In closure of the cleft in the palate I can only emphasize what Dr. Federspiel has said, that a surgical closure without functional improvement is to be regarded as a failure, and a properly made obturator will be of more benefit in these cases than an operation. I am in favor of long lateral incisions, not merely over the hard palate, but carried back and outward behind the tuberosities. These will generally give ample freeing of the palatal flaps, so that they come together without tension I have never employed any mechanical devices for the relief of tension. Dr. Federspiel states that his method overcomes the necessity for lateral incisions. I hardly see how the metal flange can be introduced without a lateral incision, or how the plate will prevent the formation of scar tissue beneath the flap which afterwards contracts. The soft tissues have to be freed from the bone whether the plates are used or not. At the same time, the excellent results obtained by Dr. Federspiel would indicate that these plates have real merit and I hope to try them out personally. One is in no position to criticise an innovation without personal trial.

In closing I wish to congratulate the essayist on his splendid presentation of the subject and the excellent results he has been able to show.

DR. JAMES SONNETT GREENE said that he was very glad to hear Dr. Federspiel lay so much stress about the status of the soft palate, the relief of tension, the lessening of cicatrices which all goes to help in eliminating in the shortening of the palate. At the Clinic, the National Hospital for Speech Disorders, where he is Director, he finds that in order to give speech to cleft palate patients it is necessary to have as much of a soft palate as it is possible to get.

The patients are mainly interested in their speech and their sole object in undergoing an operation, no matter what type of operation, is for the purpose of getting normal speech. Great difficulty is found when treating patients for normal speech, if following an operation there is no improvement in the status of the soft palate. Surgeons should bear this in mind and strive to get some sort of a rudiment of a soft palate so that function can be established.

Dr. Greene agreed with Dr. Federspiel's statements that unless one is satisfied that there is a sufficient amount of tissue to bridge over the gap, and that the soft palate can be lengthened and function be established, it is far better to advise the patient against an operation. The wearing of an obturator, no matter how well it may fit, is both a distressing and inconvenient thing, and the patient always has more or less trouble with it; yet with all this, it is more advisable to have the patient use one than to be operated on and not get sufficient soft palate tissue that is necessary for the obtaining of normal speech.

Dr. Greene said that recently he read a paper entitled "Some Mouth and Jaw Conditions Responsible for Defects in Speech" before the Section on Oral Surgery of the First District Dental Society. He thought that it would be in place to give a few extracts from that paper concerning Voice and Speech Conditions in Cleft Palate patients:

"In the first place, before operation, the voice is seriously impaired by the malformation of the mouth and nasal resonators. In addition there is a non-existence of the soft palate whose function is to check the air passing into the nasal cavities and to work in contact with the tongue in the production of the K and G sounds.

"Where the hard palate is cleft, consonants depending on the contact of the tongue and hard palate, such as T. D, N, R, L, are mutilated. The speech of these patients, as you know, is more or less affected; but what I wish to point out is that proportionately the speech or voice defect does not coincide with the size of the palatal defect; for there are small defects, small openings which greatly interfere with the production of speech, while in some large defects of the palate, even with harelip on both sides, one will find tolerable good speech without even resorting to the use of mechanical interferences.

"If one carefully tests cases of this kind there is usually found some existing conditions of the nose or throat responsible for this anomaly. An enlargement of the nose and its part; or adenoid vegetation in the nasopharynx is sufficient to produce this condition by acting as a substituting medium for the function of the missing palate, and thus making possible the formation of speech sounds.

"Operations for repair, the details of which you are already familiar, give as near a normal condition as is possible to obtain under existing circumstances.

"The question—whether the closure of the cleft remedies defective speech—can be emphatically answered in the negative; but I might add that the RESONANCE of the voice becomes more normal simply through the improved anatomical status. In nearly every case speech training is not only advisable, but is absolutely necessary.

"Following the operation of cleft palate the patient usually presents one or all of the following defects of speech:

"1. During the production of many of the consonants the familiar nasal resonance and nasal snort are heard. This is due to the lack of elasticity and mobility of the soft palate. The levator muscles cannot raise the palate with sufficient power. The soft palate ordinarily would have regulated the exit of the air through the anterior nares, but on account of its inefficiency the alae of the nose come into place and become compressed, resulting in more or less severe facial distortion. This condition is the most difficult to overcome.

"2. There is usually an omission of the consonants K and G. This is due to a lack of contact between the back of the tongue and the soft palate, because of the inelasticity of the soft palate, following operation.

"3. On account of insufficient surface being presented to the tongue the consonants L, T, D are often very indefinite and sometimes omitted. This results from lack of contact of the front of the tongue, the hard palate and the alveolar margin behind the front upper teeth.

'4. Where there is a harelip, there is difficulty in the production of the consonants B, P, M, W, due to the tensed upper lip not being able to come in close contact with the lower lip.

"5. The production of the consonant "S" is nearly always accompanied by a very unpleasant nasal snort on account of the soft palate working inefficiently. Following an operation it occasionally happens that the soft palate is too short to reach close to the posterior pharyngeal wall, consequently most cleft palate patients possess to a more or less degree a permanent rhinolalia-aperta voice, a nasal voice, even after everything possible has been done, both by operative and speech training measures.

"Besides what I have just read there are a few special points about these patients that stand out.

"Of course we nearly always get a history of heredity. Some relative, either near or remote, had a harelip or cleft palate. These patients also present neuropathic manifestations. They show peculiar traits and find difficulty in adjusting themselves to situations. The nervous system of these patients show increased irritability with diminished capacity. They are high strung, emotional, lack standardization, extremely sensitive and subject to periods of depression.

IN MEMORIAM.

HENRY LOWNDES LYNAH.

Hendy Lowndes Lynah was born in Charleston, S. C., on December 1, 1878, and died at his father's home in that city on March 31, 1922.

He was graduated from the South Carolina Medical College on April 3, 1900, and immediately afterward entered the John Hopkins in Baltimore as Extern; later becoming Intern in that institution.

His next move was to New York, where he was for a considerable time Intern at the St. Marks, Kingston Avenue and Willard Parker Hospitals. In private work he was widely known in New York for many years.

He became a member of the American Academy of Ophthalmology and Oto-Laryngology, the American Laryngological, Rhinological and Otological Society, the American Laryngological Association, the Medical Society of the State of New York, being this year Chairman of its Laryngological Section. He was one of the Founders and third President of the American Bronchoscopic Society.

Professionally he was actively engaged in the practice of his specialty at the Willard Parker and Riverside Hospitals, the Kingston Avenue Hospital and the Queensborough and Otisville Hospitals. He was Consulting Bronchoscopist and Esophagoscopist to the French, Lenox Hill, New York Eye and Ear Infirmary, the Skin and Cancer Hospital and the Methodist Episcopal Hospital of Brooklyn.

His teaching positions have been Instructor in Laryngology and Intubation at the University and Bellevue Medical College, and he was made Professor of Bronchoscopy and Esophagoscopy in the New York Polyclinic.

In the practice of the newer specialty of Bronchoscopy and Esophagoscopy he was second to only one man in this country, perhaps in the world.

In devising new instruments and methods of simplifying their application, he stood pre-eminent.

We all must contemplate with the deepest sense of loss, the removal in his prime, of this outstanding personality. The loss to the profession and the public is almost irretrievable,

and to those of his close personal friends, with whom he was in intimate contact, it is irremediable.

Realizing lately, that his career must be short, he pursued his work with cheerful confidence, not sparing himself, with a courage that must be a lesson to all who knew him well.

"So be my passing!
My task accomplished and the long day done,
My wages taken, and in my heart
Some late lark singing,
Let me be gathered to the quiet west,
The sundown, splendid and serene,
Death."

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